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EKG Interpretation

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Course Objectives

At the end of this course, each student will be able to:

1. Perform satisfactorily on an objective examination (70% or better) at the end of this course in relation to all material presented in this course.
2. Name and discuss the four most important anatomical structures of the heart that relate to the EKG.
3. Recognize one of the four most dangerous arrhythmias presented in the course, which could be dangerous to a person, including the two most frequently used treatment methods.
4. Interpret correctly the basic arrhythmias presented in this program by using the step-by-step approach outlined in the course.

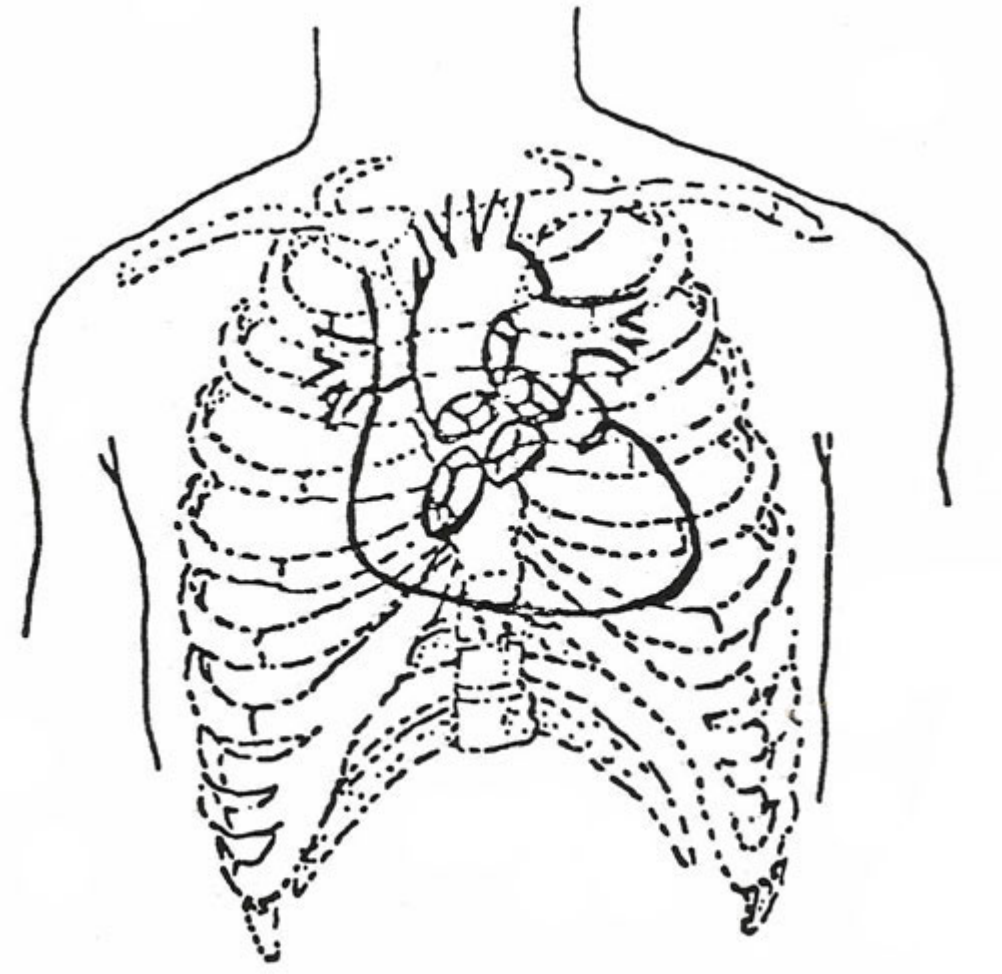
Chapter I: Physiology of the Heart and Circulatory System Campaign

Anatomy and Physiology Update

A. The Thorax

The first part of this course is a review of the anatomical structures and the physiology of the cardiovascular system in relationship to the nursing care of the patient with a related disorder. If you need to review the structures and/or function that we will be discussing, please refer to any basic anatomy textbook. However, it is not required that you use any other reference. All information needed to pass the test at then of this course will be including in the text.

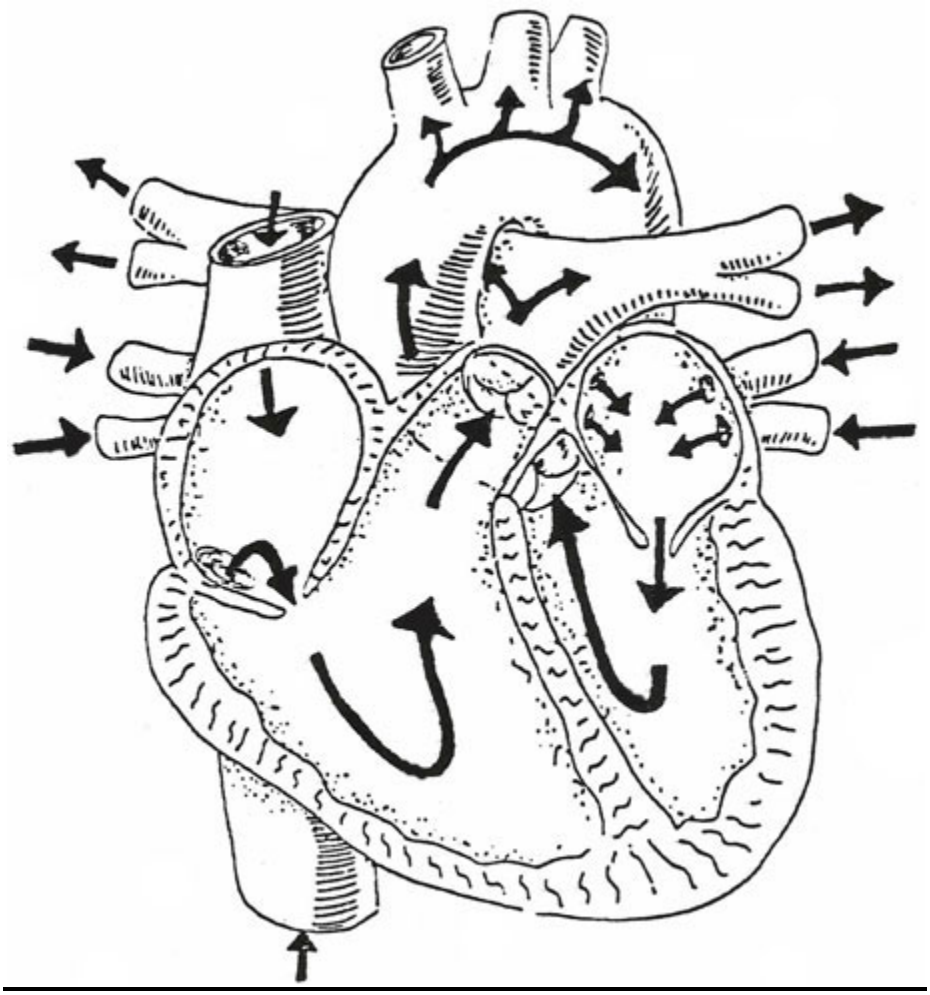
We will list the most important structures of the thorax and entire CV system only so that you can relate these to the clinical approach that we will use. Following is an illustration of the thorax and the heart in relationship to other structures noticed upon visual examination. The thorax has a characteristic shape, size and movement.



Thorax – Be sure you can identify the following:

a. Sternum – mid-chest, flat, non-protruding
b. Ribs – slope of ribs, intercostals spaces, costal margins
c. Heart – heartbeat, in some cases, can be visible as a pulsation in the thorax at lower costal margin
d. Shoulder- should be relaxed, and at a 90-degree angle, look for abnormal angles and musculature that might indicate overuse of accessory muscles
e. Neck veins – should not normally be visible
f. Clavicle – clavicular line horizontal, no protrusions during breathing
g. Respirations – normal respiration should look unlabored and comfortable

B. The Heart



Gross Structures

1. Musculature-pericardium, fibrous and serous epicardium, visceral serous pericardium, myocardium, heart muscle.
2. Muscle cell (microscopic structures) – central nucleus, sarcoplasm, sarcolemma, sarcomere, intercalated discs.

Chambers – <u>Right side of the Heart</u>

1. Right atrium – the thin-walled atrium, low relative pressure receives blood from superior and inferior vena cavae, the coronary sinus and thebesian veins, and the outflow of blood through tricuspid valve.
2. Right ventricle – relatively thing muscle wall, crescent-shaped, papillary muscles, chordate tendineae, low pressure, outflow though the pulmonic valve to the pulmonary artery.

Left Side of the Heart

1. Left atrium – thicker muscle, medium pressure of blood, inflow of blood through four pulmonary veins. Outflow is the through mitral valve.
2. Left ventricle – largest muscle mass, high pressure blood flow, papillary muscles, spring-like pump action. Outflow of blood through the aortic valve and the aorta.

Cardiac Anatomy:

The human heart is a hollow, four-chambered, muscular pump. It is the major organ in the mediastinum. The pericardium is the outermost layer of the heart. It consists of parietal and visceral layers. The pericardial sac, normally containing 5 to 20 cc of fluid, protects the myocardium and prevents friction during the pumping action of the heart.

Muscle tissue, the myocardium, makes up the walls of the heart chambers. The left ventricular myocardium is 5 to 7 times thicker than the right. The inner surface of the myocardium is lined with endocardium, as are the cardiac valves and blood vessels.

The heart is divided into chambers by intraventricular and intra-atrial septa. Fibrous tissues separate the atria from the ventricles on the right and left sides of the heart. The tricuspid and mitral valves, tighter called the atrioventricular (A-V) valves, allow for the passage of blood from the atria to the ventricles.

Heart Valves

Atrioventricular Valves:

1. Tricuspid – has three leaflets, controlled by papillary muscles; chordate tendineae.
2. Mitral valve – two cusps, controlled by papillary muscles and the chordate tendineae.

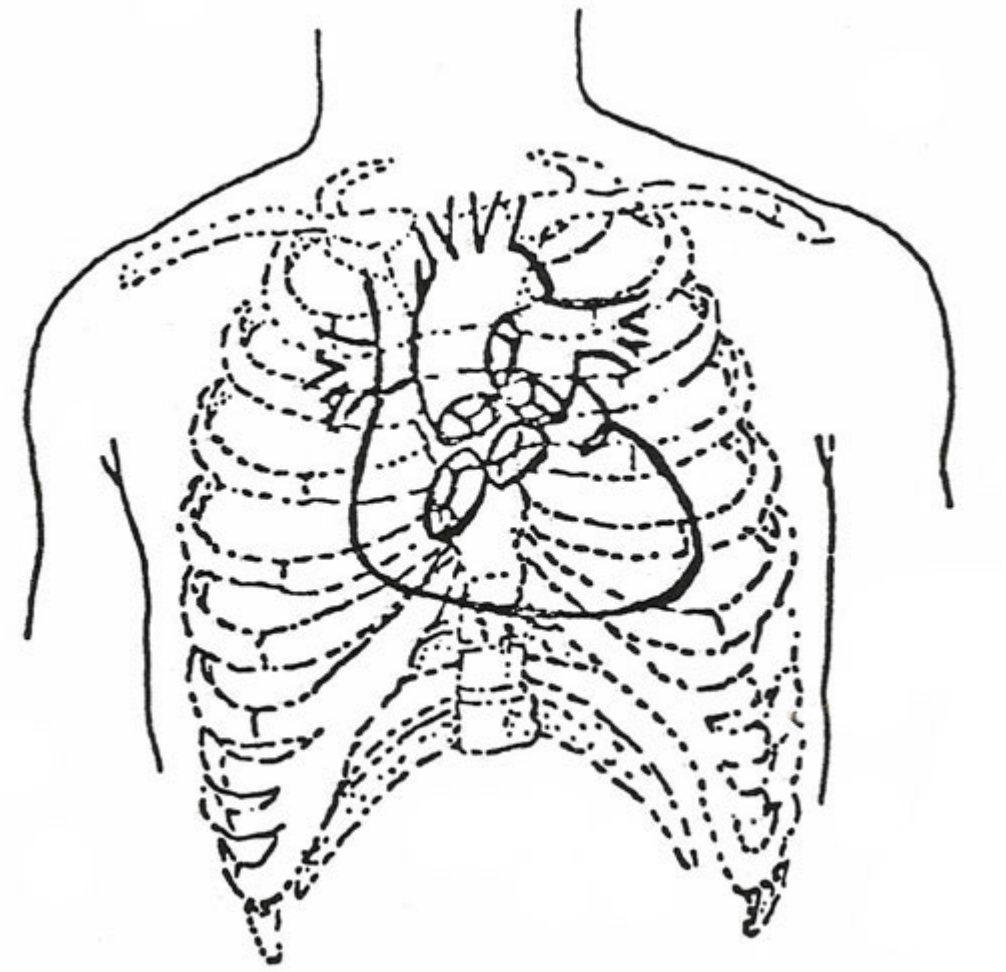
Semilunar Valves:

1. Pulmonic valve – three leaflet valve, formed by fibrous ring, tendinous tubercle midpoint free edges.
2. Aortic valve – three leaflets, also formed by fibrous ring, tendinous tubercle midpoint free edges.

Vasculature of the Heart

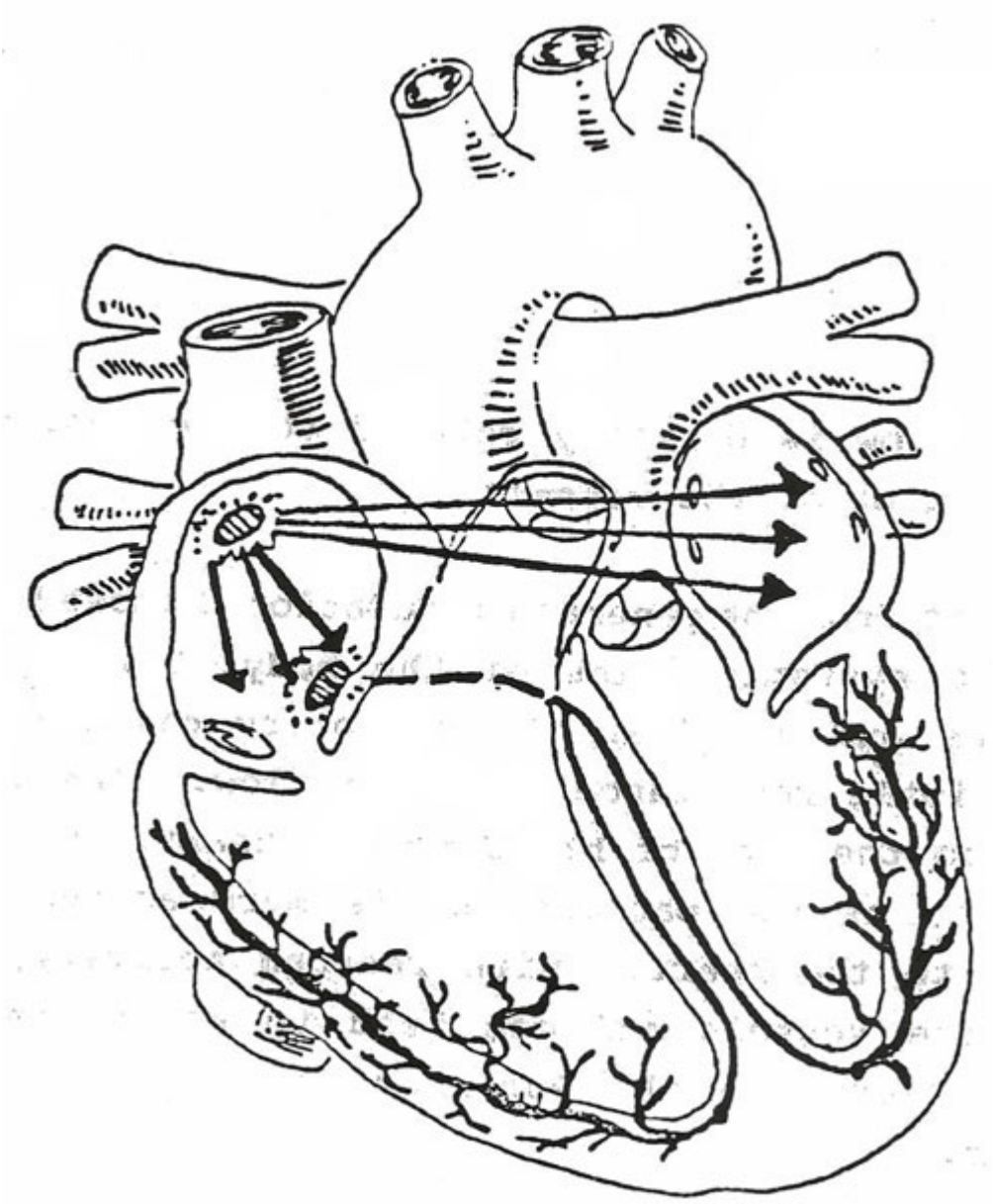
1. Right Coronary Artery – most branches of this artery anastomse distally with left anterior descending.
2. Left Coronary Artery – divides into two main branches, left ant. Descending and left circumflex artery.
3. 3. Great cardiac vein – largest system, forms coronary sinus, drains left ventricle primarily.
4. Anterior cardiac veins – empty directly into right atrium.
5. Thebesian Veins – smallest system, empty into right atrium.

Conduction System of the Heart



1. SA (Sino-atrial node)
2. Atrial preferential pathways; anterior intermodal, middle, posterior intermodal.
3. AV (Atrio-ventricular node)
4. 4. Bundle of HIS
5. Left Bundle Branch
6. Right Bundle Branch
7. Purkinje Fibres

Contractility of Heart Muscle



Electrical conduction in the heart is unique and remarkable.

Heart muscle possesses the following properties:

1. Automaticity – pacemaker ability
2. Conductivity – each cell has ability to conduct impulses to the next cell.
3. Contractility – ability to contract (make each cell shorter or longer)
4. Irritability – each call has ability to contract on its own, to send out impulses to other cells without first being stimulated from another source.

These properties make the myocardium different from other muscle cells in the body. The normal activity of the heart conducts impulses from one point (SA node) to another point (individual muscle cells) thus stimulating a uniform and effective contraction f the heart.

Various factors affect the activity of the cardiac muscles. The availability of oxygen, after load, nervous control, muscle condition, and other factors can affect the force of the contraction of the heart. Drugs can also affect the contraction of the heat. Certain drugs depress the heart actively and others can cause excitation. The nurse should be aware of all the factors that influence heart activity.

Blood Flow Through the Heart (refer back to fig. 1)

Blood flow through the heart is shown in the illustration. Blood is shown as it enters the heart, circulates, and then leaves the heart. In relation to the psychical assessment performed by most nurses, keep in mind the changes in circulation which will be assessed. Impeded flow my cause extra heart sounds and/or physical changes. Also, reduced flow will usually cause changes that can be assessed by the nurse.

Physical Characteristics Important to Blood Flow:

1. Diameter of the blood vessels.
2. Cross-section areas of the chambers and vessels.
3. Length of the vessels.

Qualities of blood:

1. Heart	18%
2. Pulmonary vessels	12^
3. Large Arteries	8%
4. Small Arteries	5%
5. Arterioles	2%
6. Capillaries	5%
7. Small Veins	25%
8. Large Veins	25%

Velocities of Blood Flow:

The velocity of blood flow is directly related to the amount of circulating blood volume and the area of the vessels.

Blood returns to the heart from the general circulation. Almost 50% of all blood in the body is in the systemic veins of the body. This system includes small veins and venules and blood in the pulmonary circulation. The small veins usually offer little resistance to blood flow. The large veins do offer much resistance to the flow of blood to the heart. This is an important nursing implication, as the patient who is more active will have better flow of blood back to the heart. With reduced activity, the blood tends to pool I the large vessels and can lead to severe venous

stasis. Blood returns to the heart via the superior and inferior vena cavae, and into the right atrium.

From the right atrium blood flows to the right ventricle and is then propelled into pulmonary circulation. After blood is aerated with fresh oxygen, it is returned to the left side of the heart into the left atrium.

From the left atrium the blood is ejected into the left ventricle. The left ventricle then pumps the blood out of the heart into the general circulation. The aorta is the first vessel to carry blood, and, at that same time, coronary arteries are fed oxygenated blood to circulate though the heart.

The above is only a brief outline of the circulation of blood. Be sure you can trace the blood through the heart. Be sure that you can name all the valves and chambers of the heart as blood flows through. You should also be able to list the major arteries of the body. When you perform the assessment, it will be necessary for you to know these vessels and their location.

Myocardium:
Following is a review of the physiology of muscle contraction.

Gross Structures:

1. Myocardium – heart muscle
2. Epicardium- visceral serous pericardium
3. Pericardium- parietal and visceral layers
4. Muscle cells
    a. Central nucleus of cell
    b. Sarcoplasm – proteinaceous fluid
    c. Sarcolemma – cell membrane
    d. Sarcomere – contractile until of muscle

Muscle contraction is dependent upon the availability of calcium and other electrolytes. The nervous system sends impulses to the muscle cells. These impulses stimulate release of calcium (Ca+) in the muscle cells. Calcium forms a link between ATP and ADP (energy sources) to initiate the contraction of the muscle cell. As these energy bonds are formed with in the muscle cell, the sarcomere is shortened. Shortening of the sarcomere causes shortening of the muscle fiber (cell), and hence the contraction of the entire muscle. When the calcium in the cell is used up, the sarcomere returns to its normal length and so does the muscle. This is known as diastole. The muscle must replenish its calcium and energy. Cardiac muscle has special properties that not all other muscles have. As mentioned earlier in the text, the properties are:

1. Contractility: the action of muscle fibers to shorten in length (contraction)
2. Conductivity: each muscle cell can pass electrical impulses from cell to cell.
3. Automaticity: ability to contract without direct stimulation by nervous system
4. Irritability: ability to respond in a specific way to changing conditions of body tissues.

Muscle Function

In the body many conditions must be met before a muscle will have the ability to contract. We know that fluids and electrolytes are important as well as the condition of the body.

Listed below are other factors that must be considered:

1. Overall muscle condition.
2. The availability of oxygen to the muscle.
3. Afterload conditions.
4. Nervous control of the muscle.
5. Electrical activation of the muscle.
6. *Frank Starling Law*: the longer the muscle is stretched during diastole, to a point, the stronger the contraction in the next systole.
7. Cardiac output can be increased by the increased availability of sites for electrochemical bonding when the sarcomere is stretched, up to a point.
8. Decreased end-diastolic volume: (hemorrhage, dehydration, etc.) sarcomeres too short to perform properly and cardiac output decreased.
9. All or none principle: the nervous innervations must reach a threshold strong enough to trigger the muscle; even with this minimum nerve impulse, the muscle will contract to its full potential.

The preceding material is review of the anatomy and physiology of muscle contraction. If this review "jogs" your memory, then the purpose was accomplished. It is important that you keep in mind the basic principles of muscle physiology. These principles will help you understand some of the following normal and abnormal ECG tracings.

Anatomy Related to the ECG

Represented next is the electrical pathway of the impulses through the heart. Each wave on the ECG is related to a portion of those impulses. When the heart muscle is stimulated by the electoral impulses, blood is ejected from eh corresponding chamber of the heart.

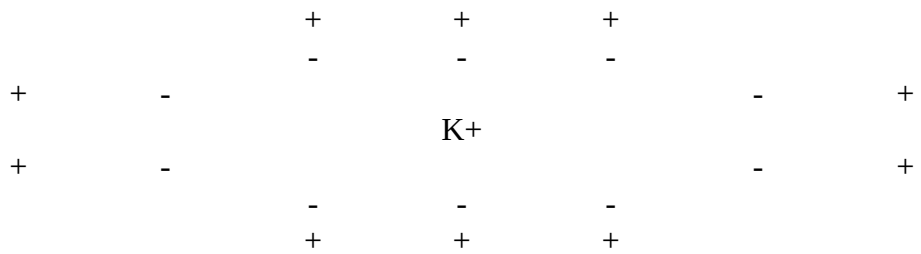
a. Basic Facts:
    1. Fluid bathes the inside and outside of the cell membrane.
    2. The fluid is an electrolyte solution carrying (+) and (-) ions.
    3. Current will flow between ions of opposite polarity.
    4. When cells are at rest, the extracellular fluid is mostly positive, therefore, there will be no current flow.
    5. When the cell membrane is stimulated, current will flow.
    6. K+ goes in and out freely at all times.
    7. CL- is equal on both sides of the cell.
    8. K+ is limited outside the cell.
    9. Process is diffusion gradient change and the negative chargers inside the cell attract NA+ ion to enter cell's interior.

b. Cellular Physiology Terminology
    1. **Vector** - Symbolic representation of physical force.
    2. **Electric Tension** - Latent energy resulting from the collection of (+) and (-) ions.
    3. **Resting Membrane Potential** – (polarized state) in a polarized cell there are an equal number of (=) and (-) chargers. Normal resting membrane potential is -85mv to -95mv.
    4. **Depolarization** - is when the polarized state has been interrupted by a stimulus, the result is that Na+ ions rush inside the cell and some K+ leaves the cell, and the cell's polarity changes.
    5. **Repolarization** - is the state where the cell returns to its polarized state and the polarity reverses.
    6. **Action potential** - the rapid sequence of depolarization and repolarization.
    7. **Selective Permeability** - with all living cells, the membrane passes some substance but blocks others.
    8. **Sodium pump** - a metabolic pump that pumps Na+ out of a cell, and pumps K+ to cell's interior, this occurs only in diastole.

C. Cellular Activity

1. Resting Cell
    a. Has equal number of (+) and (-) charges.
    b. Repolarized.

    Example:



2. Depolarized Cell
    a. Exchange of Na+ and K+ and polarity reverses

3. Repolarized Cell
    a. Reversal of polarity and exchange of Na+ and K+

K+

(+) Na+

D. Uniqueness of Pacemaker Cell vs. Non-Pacemaker Cell
1. SA Node
Pacemaker cells have one phase of action potential. The reason is because of a time – dependent decay in K+ conductance. This, plus passive diffusion of Na+ back into the cell, causes a threshold to be reached. (Constant Na+ leakage, and constant K+ moving) All action is self-initiated.

2. Phases of Action Potential:

Phase 0

This is the portion from the threshold to peak action potential (i.e. the exchange of Na+ and K+ in the cells).

Phase 1

Initiate stage of repolarization, it has a brief origin (initial phase due to influx of C1- ion).

Phase 2

During the next 0.1 second, the repolarization process slows. This period does not exist in skeletal muscle. This allows cardiac muscle to have a more sustained contraction (isometric contraction; plateau phase).

Phase 3

Sudden acceleration of the rate of repolarization as K+ leaves the cell in response to the influx of Na+.

Phase 4

This represents the resting potential.

E. Membrane Responsiveness

1. Definition
The relationship of resting membrane potential at excitation to the rate of depolarization during phase 0 of the action potential; normal resting membrane potential is 85 to 90 mv.

Maintenance of Membrane Potential

Is dependent upon the integrity of the cell membrane; injury, ischemia, chemical intoxication and also radical temperature changes can alter membrane potential.

F. Refractory Periods
1. Relative Refractory Period

Some cells are polarized and others are not. A strong stimulus can produce a response in the polarized cells (after P wave complex by T wave).

2. Absolute Refractory Period

When cells are refractory and unable to accept any stimulus.

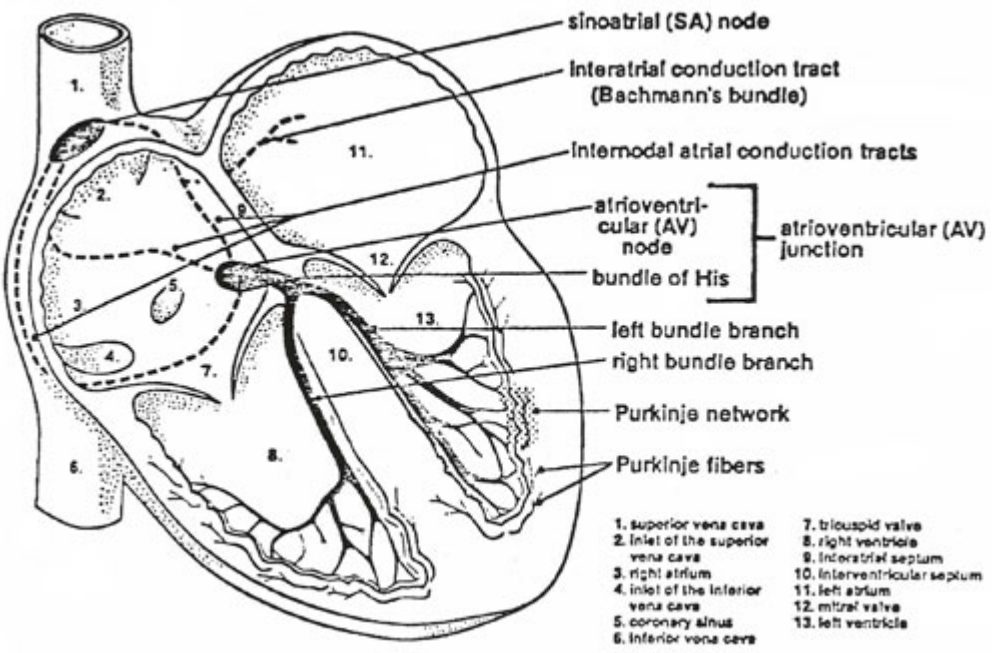
G. Vectors
A vector is a symbolic representation of a physical force. It has direction and magnitude, characterized by an arrow, (Plus or Minus). Vector size varies; it depends upon muscle mass.

H. Axis

An axis is a center point or center line (symbolic). Normal flow of current is in the heart is primarily from the base to the apex. This preponderant flow of current is known as the axis. Axis is the sum total of all the small vectors.

I. Conduction System Components

Electrical Conduction System of the Heart:



The electrical conduction system of the heart is composed of sinoatrial (SA) node, internodal atrial conduction tracts, interatrial conduction tract, atrioventicular (AV) node, bundle of HIS, right and left bundle branches, and Purkinje network. The AV node and the bundle of HIS form the AV junction. The bundle of HIS, the right and left bundle branches, and the Purkinje network are also known as the HIS-Purkinje system of the ventricles. As its sole function, the electrical conduction system of the heart transmits minute electrical impulses from the SA node (where they are normally generated) to the atria and ventricles, causing them to contract.

The SA node lies in the wall of the right atrium near the inlet of the superior vena cava and consists of pacemaker cells that generate electrical impulses automatically and regularly. The AV node lies partly in the right side of the interatrial septum in front of the opening of the coronary sinus tricuspid valve.

Electrical Basis of the EKG (Based on the pathway depicted in the figure above.)

The electrocardiogram, ECG, is a graphic record of the direction and magnitude of the electrical activity that is generated by the depolarization and repolarization of the atria and ventricles. This electrical activity is readily detected by the electrodes attached to the skin. In the next section we will go into more detail about the EKG tracing and the normal EKG. This will lay a foundation for interpretation of arrhythmias to come later.

We use EKG – ECG interchangeably. They mean the same thing. EKG was a term originated from old English, and is still used today. Some facilities prefer one term over the other. Use the term that is preferred at your facility.

Chapter II: The Electrocardiogram and the Normal EKG

Part I: The ECG Paper and the Normal ECG Tracing

In order to begin to understand the interpretation of EKC's one must have an understanding of the EKG paper. Shown in the illustration below are a sample of EKG paper and an enlargement of the markings. The time intervals are shown as well as the measurements of each block on the paper. You will be able to make fairly accurate measurements of the patient's heart rate and other measurements by counting blocks up and down on the paper.

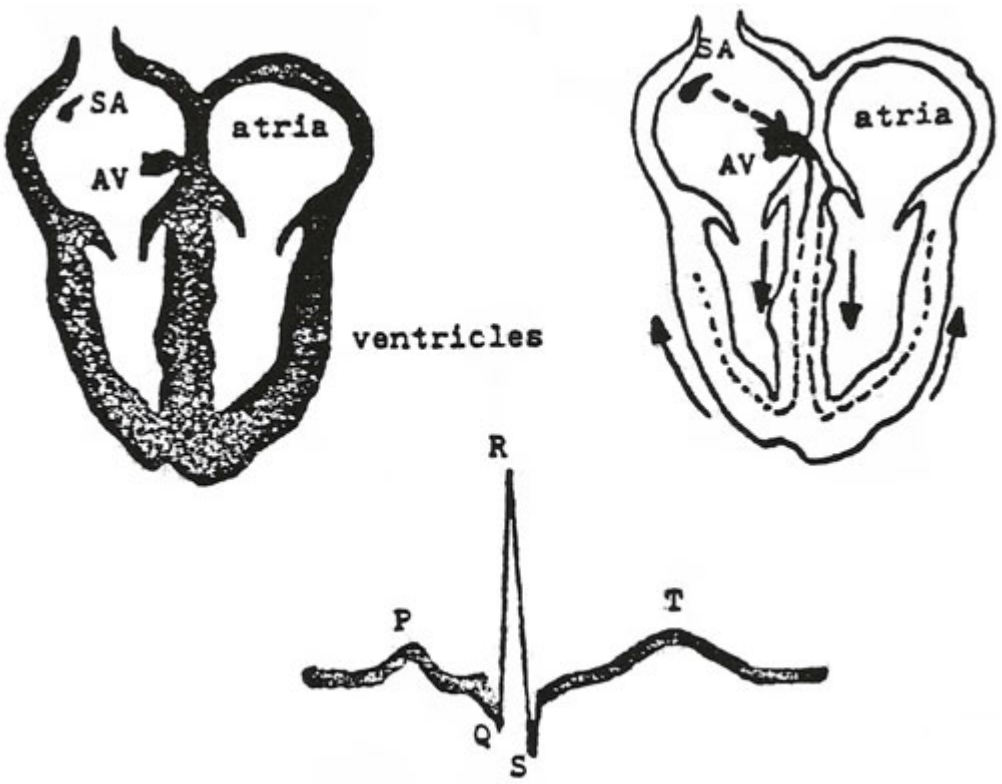
The EKG paper records time sequences (horizontal deflections) and amplitude (vertical deflections) of the electrical activity of the heart. The horizontal lines measure time intervals and heart rate. Each of the small squares equal 0.4 second of time. Five small squares equal 0.20 seconds. Fifteen of the 0.20 squares represent 3 seconds. These 3 – second time intervals are marked on the paper by darker lines as shown below.

Conversion table for heart Rate

Listed here is a quick guide for determination of heart rate:

Number of Small Spaces:	Rate Per Minute
30	50
29	52
28	54
27	56
26	58
25	60
24	63
23	65
22	68
21	72
20	75
19	79
18	84
17	88
16	94
15	100
14	107
13	115
12	125
11	136
10	150
9	167
8	188
7	214
6	250
5	300

Components of Normal Sinus Rhythm



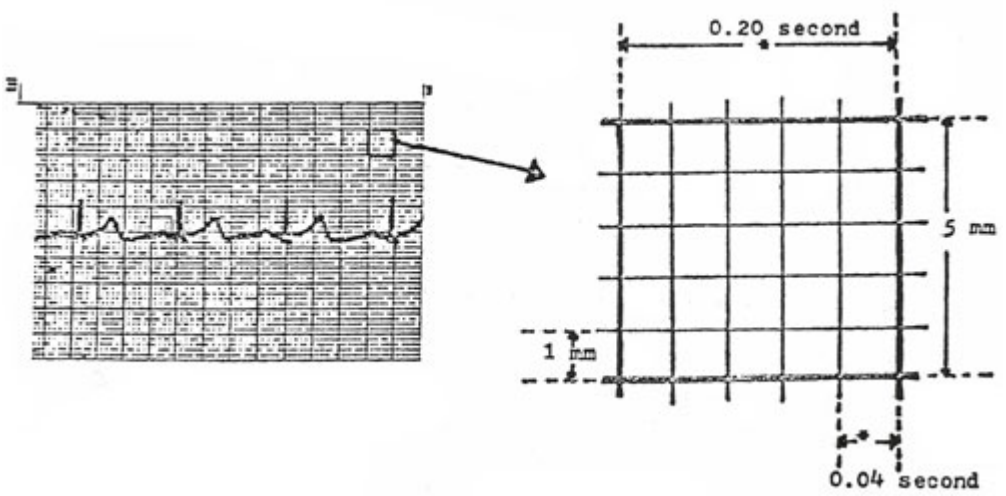
P Wave:

The P wave represents depolarization of the right and left atria.

Onset of the P Wave is identified as: the first abrupt or gradual deviation from the baseline. The point where the wave returns to the baseline marks the end of the P Wave. A QRS complex normally follows each P Wave. A normal sinus P Wave indicates that the electrical impulse responsible for the P Wave originated in the SA node and that normal depolarization of the right and left atria has occurred.

1. SA node is the pacemaker site.
2. The first part of the normal sinus P Wave represents depolarization of the right atrium; the second part represents depolarization of the left atrium.
3. During the P Wave, the electrical impulses progress from the SA node through the intermodal atrial conduction tracts and most of the AV node.
4. The DIRECTION of the P Wave in lead II is positive (upright).
5. The DURATION is 0.10 seconds or less.
6. The AMPLITUDE is 0.5 to 2.5 mm high, in lead II (rarely over 2mm high)
7. The SHAPE is normally smooth and rounded.
8. The P-R INTERVAL is normally 0.12 to 0.20 seconds; abnormal is greater than 0.20 seconds.

QRS Complex



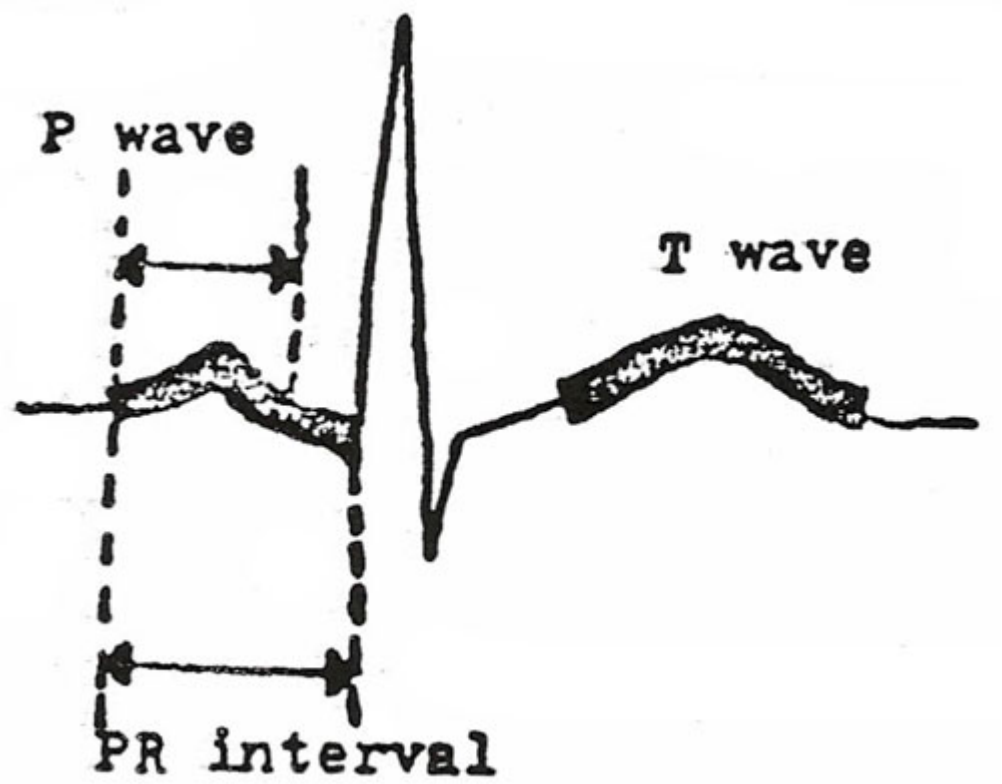
A QRS Complex represents depolarization of the right and left ventricles.

The pacemaker site of a normal QRS complex is the SA node or an ectopic pacemaker in the atria of AV junction. (The origin of the QRS is originally from the SA node, and then spreads down through the atria to the AV node, etc.) The onset of the QRS Complex is identified as the point where the first wave of the complex just begin to deviate, abruptly or gradually, from the baseline.

This end of the QRS Complex is the pone there the last wave of the complex begin to flatten out, sharply or gradually, at, above, or below the baseline. This point the junction between the QRS complex and the S-T segment is called the "JUNCTION" or "J POINT." A normal QRS Complex indicates that the electrical impulse has progressed normally from the bundle of HIS to the Purkinje network through the right and left bundle branches, and that normal depolarization of the right and left ventricles has occurred. Of course, there can be several "normal" variations of the QRS Complex. These will be discussed later in the course.

1. The Q Wave is the first negative deflection in the QRS Complex not preceded by an R Wave.
2. The R Wave is the first positive defection in the QRS Complex.
3. The S Wave is the first negative deflection that extends below the baseline in the QRS Complex that follows the R Wave.
4. The DURATION of the QRS Complex is 0.06 to 0.10 seconds in adults and 0.08 or less in children.

T Wave



A T Wave represents ventricular repolarization.

Repolarization of the ventricles begins at the epicardial surface of the ventricles and progresses inwardly through the ventricular walls to the endocardial surface. The T Wave occurs during the last part of the ventricular systole. The onset of the T Wave is the first or abrupt or gradual deviation from the S-T segment; or from the point where the slope of the S-T segment appears to become abruptly of gradually steeper. If the S-T segment is absent, the T Wave begins at the end of the QRS Complete (or the J Point). The point where the T Wave returns to the baseline marks the end of the T Wave. Often the onset and end of the T Wave are difficult to determine with certainty.

1. The DURATION of the T Wave is 0.10 to 0.25 seconds or greater.
2. The AMPLITUDE of the T Wave is less than 5 mm.
3. The SHAPE of the T Wave is sharply or bluntly rounded and slightly asymmetrical.
4. A T Wave always follows a QRS Complex.

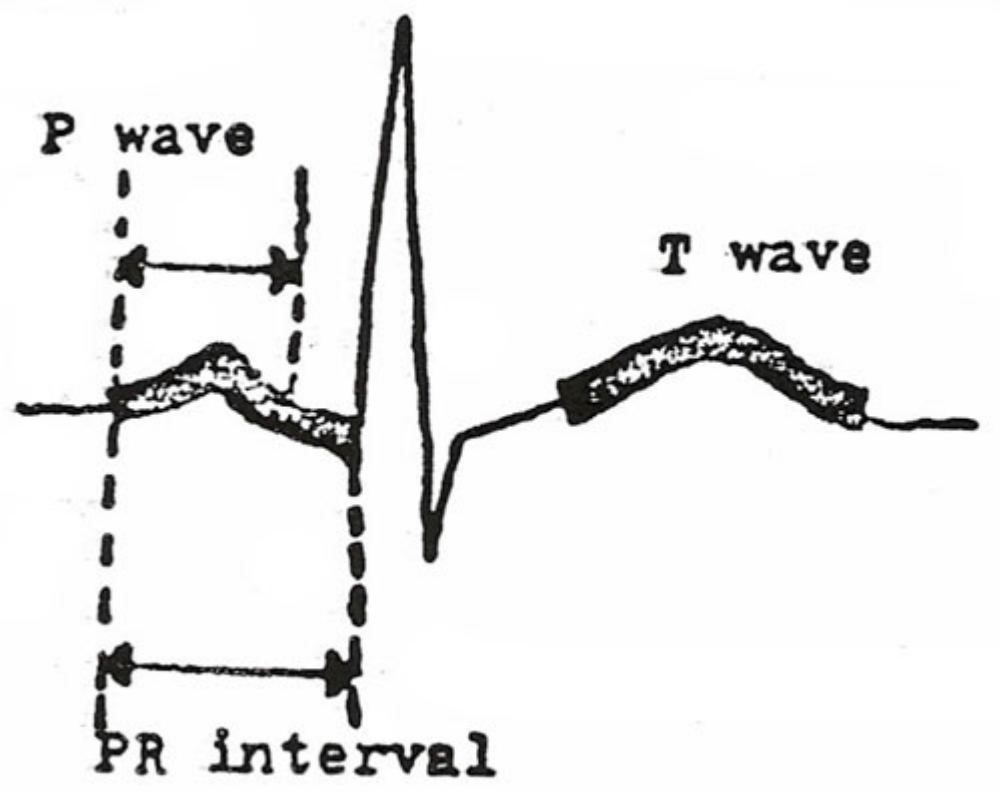
The U Wave

The U Wave probably represents the final state of repolarization of the ventricles.

The U Wave probably represents repolarization of a small segment of the ventricles, such as the papillary muscles or ventricular septum, after most of the right and left ventricles have been repolarized. Although uncommon, and not easily identified, the U Wave can best be seen when

the heart rate is slow. A U Wave indicates that the repolarization of the ventricles has occurred. An abnormally tall U wave may be present in hypokalemia, cardiomyopathy, left ventricle hypertrophy, diabetes, and may follow administration of digitalis and quinidine.

1. The ONSET of the U wave is identified as the first abrupt or gradual deviation from the baseline or the downward slope of the T Wave.
2. The END of the U Wave is the point where it returns to the baseline or downward slope of the P Wave.
3. The DIRECTION of the U Wave is positive (upright), the same as that of the preceding normal T Wave in lead II. An abnormal U Wave may be flat or inverted.
4. The DURATION of the U Wave is normally not determined and the duration is normally not significant, except in rare cases.
5. The AMPLITUDE of a normal U Wave is usually less than 2mm and always smaller than that of the preceding T Wave in lead II.
6. The U Wave always FOLLOWS the peak of the T Wave and occurs before the next P Wave.



The EKG Leads

The 12-lead EKG will be discussed in greater detail later in this course. However, at this time we will present an introduction to the EKG leads simply to help explain the basics of EKG interpretation that will follow. Later, the specifics of leads and lead placement will be discussed.

An EKG lead consists of two surface electrodes of opposite polarity (positive and negative) or one positive surface electrode and one reference point. A lead composed of two electrodes of

opposite polarity is called a Bipolar Lead. A lead composed of a single positive electrode and a reference point is called a Unipolar Lead.

All leads of the ECG record the same electrical impulses of the heart muscle. However, each lead placed in a different area of the body, records the electrical activity from a slightly different "angle." This means that by using the ECG tracing from different positions of the chest, various ECG waves will be accentuated. Diagnosis of arrhythmias may be made easier by examination of different leads. The 12-lead ECG tracing is standard. Six leads are recorded by placing wires on each limb. The other six leads are recorded by placing wires on the chest in six specific positions.

Limb Leads: I, II, III, IV, V, VI

Lead IV also called AVR
Lead V also called AVL
Lead VI also called AVF

Chest Leads: V_1, V_2, V_3, V_4, V_5, V_6

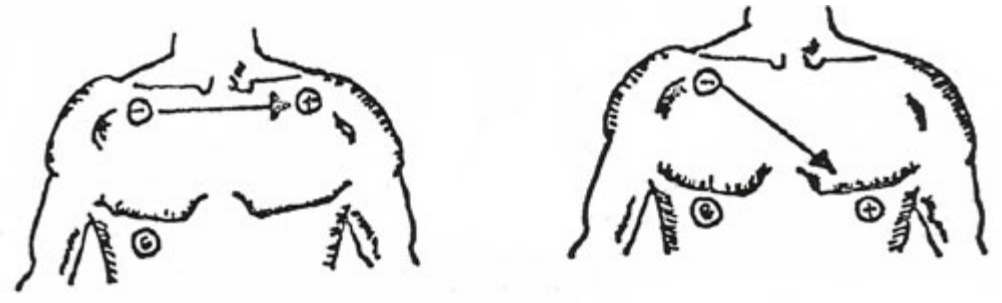
For diagnosis of most arrhythmias, lead II is most commonly used. Lead II (and the chest leads) most consistently show the most clear P Wave which can be diagnostic of many common arrhythmias.

The following leads are listed and their relationship to areas of the heart muscle:

V_1, AVR	Right side of the heart.
V_2, V_3, V_4	Transition between right and left sides of the heart.
V_5, V_6, I, AVL	Left side of the heart.
II, III, AVF	Inferior aspect of the heart.

If changes in the ECG tracing are seen in a group of the above leads, the disease can be localized to a particular area of the heart. In the case of an MI which show changes in the leads V_1 and AVR only, the damage to the heart is in the right side. If the MD can thus localize the damage to the heart they can also diagnose other possible problems in the heart. Valvular problems may show up as a specific change in one or more leads of the ECG tracing. Blockages in one of the major arteries of veins may also show up as an altered deflection in the ECG.

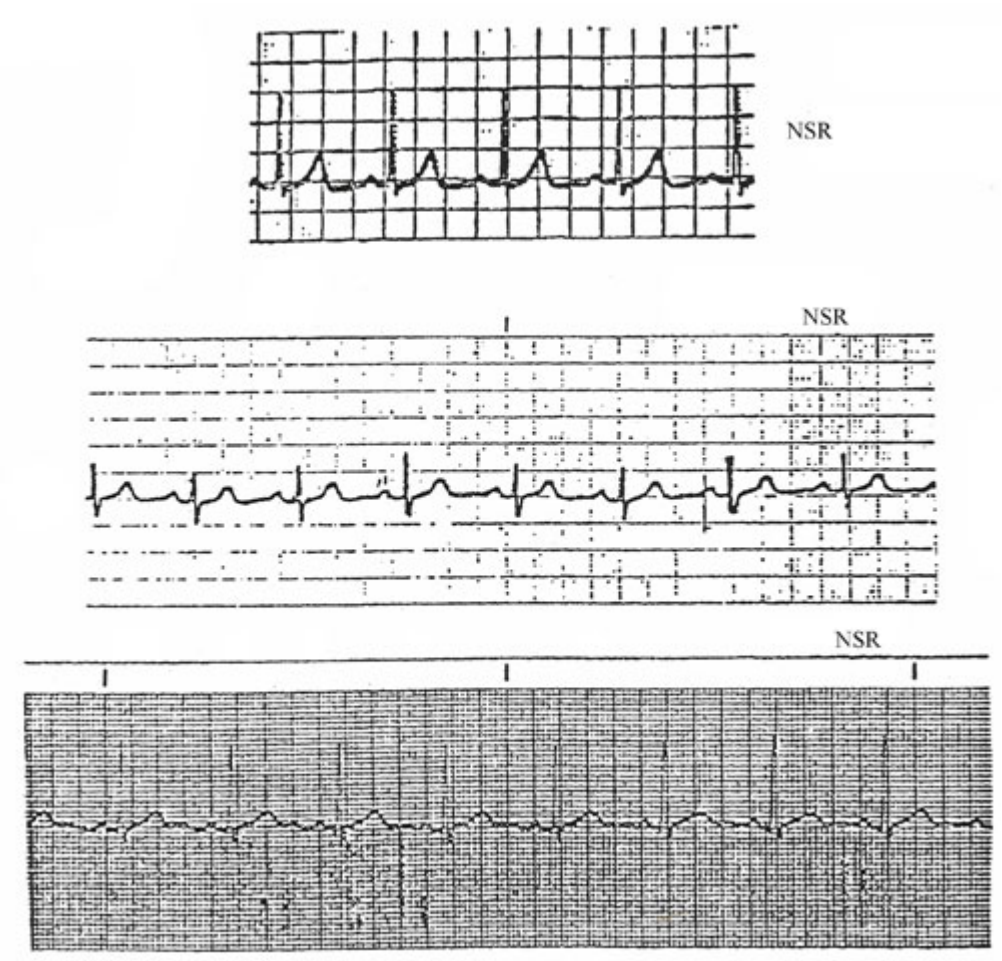
Below is a diagram of the chest and the placement of leads of the chest so as to trace leads I and II. When the patient is being monitored for a specific arrhythmia, it will help to connect the wires to the spot of the chest that will show that arrhythmia most clearly.



G = Ground Point (Reference Point)

Normal Sinus Rhythm

Normal Sinus Rhythm, NSR, is the term used to describe the normal heartbeat. Looking at the EKG tracing, all beats appear similar and are evenly spaced. The NSR implies that all of the beats have a normal pacemaker (starts at the SA node). All of the beats also follow the normal conduction pathways in NSR. Lastly, all components of the waves are similar, the P Wave, QRS Complex, and T Wavers are similar to each other.



Arrhythmias

The normal EKG consists of repetitive series of P, Q, R, S, and T Waves, which conform to established standards for size and shape and occur 60 – 100 times each minute. If these conditions prevail, the heart is in normal sinus rhythm. When either the rate of the contour of any of the individual waves is abnormal, the disorder is called an arrhythmia.

Classifications of Arrhythmias:

There are several ways in which arrhythmias may be classified. Perhaps the most logical method involves classification, first, by the site of the arrhythmia and secondly by the type of mechanism responsible for the disorder.

Sites	Mechanisms
SA Node (sinus rhythms)	Tachycardia (rate over 100 bpm)
Atrial (atrial rhythms)	Bradycardia (rate under 60 bpm)
AV node (nodal rhythms)	Premature beats
Ventricles (ventricular rhythms)	Flutter Fibrillation Defects in conduction.

Arrhythmias can also be classified in a general way according to their seriousness or prognosis. This is a meaningful method for nurses caring for patients with acute Myocardial Infarction and the one we have found useful.

Using this division, arrhythmias may be considered as:

1. **Minor** – these arrhythmia are not of the immediate concern and generally do not affect circulation. They are important because they frequently reflect irritability of the heart.
2. **Major** – these disturbances reduce the efficiency of the heart of warn of impending danger and require prompt treatment.
3. **Death Producing** – Immediate resuscitation is needed to prevent death.

Identifying ECG Features – Sinus Arrhythmias

1. **Rate** (In Sinus Arrhythmias)
   The heart rate per minute is normal (60-100); however, the rate increased during inspiration and then slows during expiration.
2. **Rhythm** (In Sinus Arrhythmias)
   Irregular rhythm; there is a variation of at least 0.12 seconds between the longest and shortest R-R intervals.
3. **P Waves** (In Sinus Arrhythmias)
   P Waves are normal.
4. **PR Interval** (In Sinus Arrhythmias)
   Normal; each P Wave is followed by a normal QRS complex.

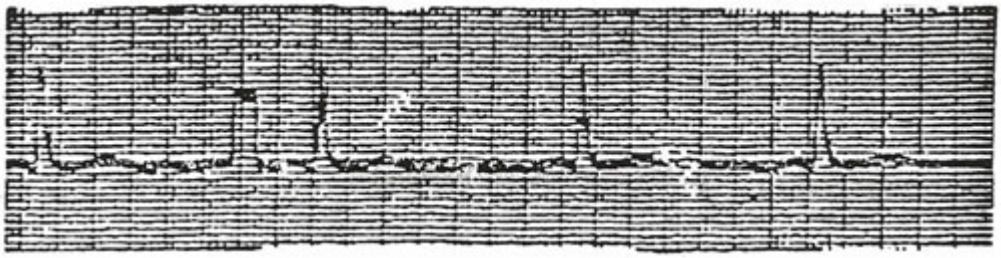
5. **QRS** (In Sinus Arrhythmias)
   QRS is normal width

Sinus Arrhythmia:

Rate:	About 80/minute.
Rhythm:	There is a variation in R-R intervals of more than 0.12 seconds.
P Waves:	Normal
PR Interval:	Normal (0.12 second).
QRS:	Normal (0.04 second).
Comments:	The short R-R intervals occur during inspiration and the long R-R intervals during expiration.

Sinus Bradycardia

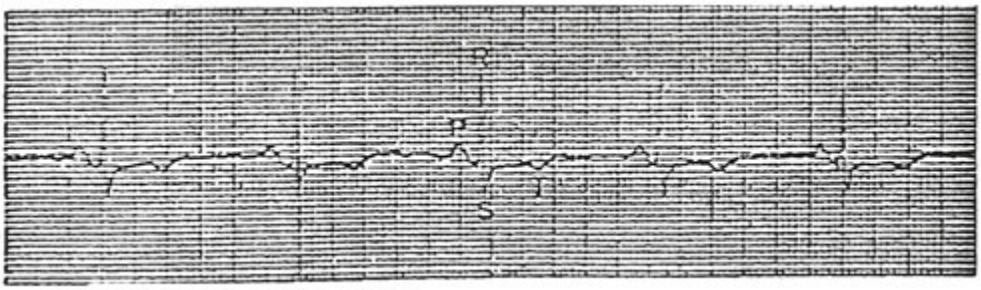
Sinus Bradycardia is an arrhythmia defined as a rate below 60 BPM with all beats remaining normal. The SA node is still the pacemaker and the conduction pathway is still normal. This rhythm can be normal during sleep, for athletes, for persons on certain medications and other reasons. It can be an abnormal rhythm in certain conditions such as myocardial infarction or congestive heart failure, or if the rate falls so low as to cause clinical symptoms such as fainting of dizziness, etc.



Sinus Bradycardia Indentifying ECG Features

Rate:	Usually 40 – 50 /minute, but may be slower.
Rhythm:	Regular.
P. Waves:	Normal.
PR Interval:	Normal and each P wave are followed by a normal QRS complex.
QRS:	Normal

Example Sinus Bradycardia



INTERPRETATION OF ECG

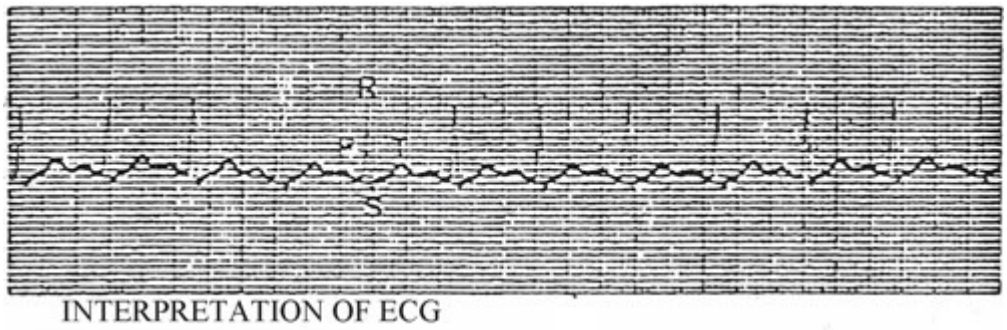
Rate:	About 50/minute
Rhythm:	Regular.
P Waves:	Normal.
PR Interval:	Normal (0.16 second).
QRS:	Normal (0.08 second).
Comments:	The inverted T waves are not related to the arrhythmia; they result from myocardial ischemia.

Sinus Tachycardia

Sinus tachycardia is another sinus arrhythmia. Causes of this condition are exercise, anxiety, fear, fever, and others. If the tachycardia is secondary to another factor, such as above, usually no treatment is needed, as these conditions are usually temporary. However, if clinical signs are seen, such as dizziness, fainting, or others, treatment may be indicated and perhaps more than just simple sinus tachycardia is present.

Sinus Tachycardia Identifying ECG Features	
Rate:	Usually 100-150/minute.
Rhythm:	Regular.
P Waves:	Normal. (If the rate is very rapid, the P waves may not be clearly identified because they may encroach on the preceding T Waves.)
PR Interval:	Normal, indicating that conduction from the SA node through the ventricles is not disturbed.
QRS:	Normal

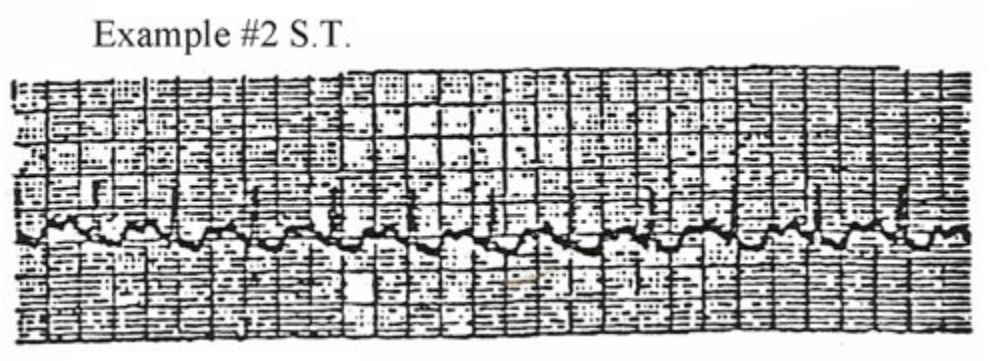
Example: Sinus Tachycardia



INTERPRETATION OF ECG

Interpretation of ECG	
Rate:	About 120/minute
Rhythm:	Regular.
P Waves:	Normal.
PR Interval:	Normal (0.16 second), and each P wave is followed by a normal QRS complex.
QRS:	Normal (width is 0.06 second).
Comments:	Other than the rapid rate there are no abnormalities.

Example 2 Sinus Tachycardia



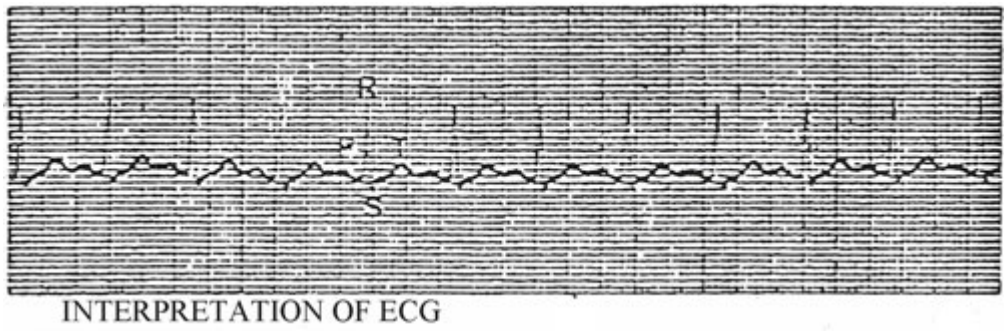
Part II: Abnormal Rhythms

A. Atrial Arrhythmias

1. Atrial Tachycardia and Paroxysmal Atrial Tachycardia (PAT)

PAT is a relatively common arrhythmia in young adults and usually means no permanent heart damage. Many young adults experience brief episodes of "fluttering" or "pounding" in the chest that also may cause a short period of weakness. The episode may be so brief that it goes almost unnoticed.

On the ECG, PAT will be seen as the heart beating at a rate of 160 to 240 Beats per Minute (BPM). The P Wave will be shaped differently than the normal P wave. When the impulses from the SA node travel the normal pathway to the AV node, the ECG shows a "normal P wave." However, when PAT is present, the pacemaker is not the normal SA node. The P wave has a different shape due to this fact.

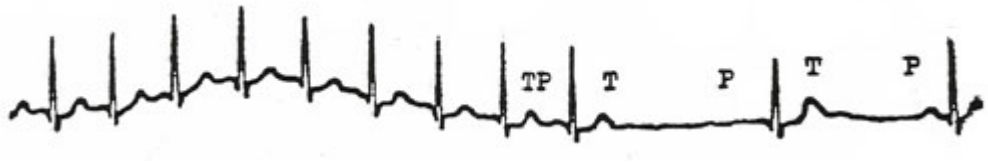


INTERPRETATION OF ECG

By definition, PAT commonly starts and ends abruptly (in paroxysms). Atrial Tachycardia is defined as three or more consecutive (abnormal) atrial contractions. Atrial Tachycardia then, is sustained abnormal atrial contractions. Therefore, PAT is defined as just an occasional abnormal atrial contraction (fewer than three in a row).

Supraventricular Tachycardia is a term used to indicate a paroxysmal tachycardia originating in the atria of AV junction without specifying the exact location of the ectopic pacemaker site.

Many times is difficult to determine the site so the arrhythmia is called supraventricular tachycardia.



As you see from the previous PAT example, there is an abnormal pacemaker that sends impulses to the AV node. The ventricular wave (QRS) appears normal but rapid in response to the rapid atrial stimulation. To summarize, PAT is recognized by a rate of over 140 per minute (higher than sinus tachycardia), normal QRS complexes and abnormally shaped P Waves when they are visible and not hidden by the preceding T Wave.

Treatment of PAT

As we mentioned earlier, PAT will often stop spontaneously and requires no treatment. IN more severe cases, which are either prolonged or case clinical symptoms, treatment is required. In these severe cases, the ECG machine will remain attached to the patient because many of the treatments for PAT can in themselves cause cardiac complications. The simplest form of treatment may be the administration of a sedative or tranquilizer. In ten to fifteen minutes, the episode may terminate. This procedure stimulates the heart-slowing vagus nerve and may break the episode of PAT. Gagging the patient with a tongue depressor may accomplish the same thing.

In cases where none of the above drugs may be used due to the patient's condition, cardioversion (administering a synchronized electric shock) may be useful. The shock delivered stops the heart momentarily and then allows the normal pacemaker of the heart to take over. For most cases of PAT, however, the drug therapy with Aramine works quite well.

2. **Atrial Flutter**

This arrhythmia is similar to PAT in origin. The pacemaker for flutter is also located on one spot in the atrium, usually in the lower atrium near the AV node. The rate for flutter, however, is faster than PAT, 250-350 per minute. Another difference with flutter is that not all of the P waves cause conduction of the ventricles and subsequent contraction. In PAT each rapid P wave caused ventricular responses, there was no blockage of the electrical impulses. However, with atrial flutter, P waves come too fast for each of them to cause a ventricular contraction.

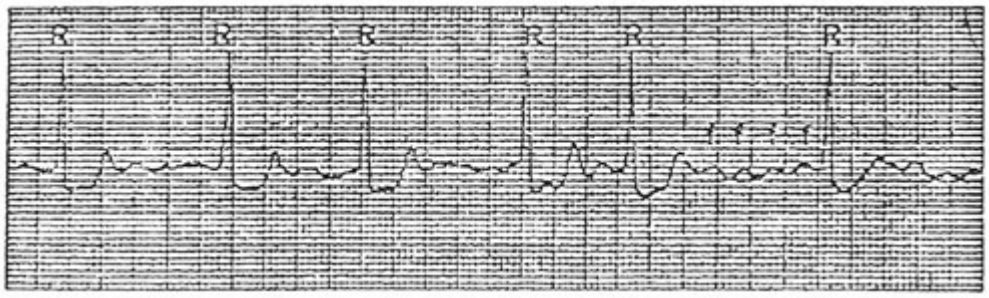
Atrial flutter is usually treated by digitalization. The digitalis slows the pulse rate by blocking the impulses at the AV node so that fewer impulses get through to the ventricles. Some persons with this arrhythmia have no clinical symptoms. Other persons become weak and dizzy, and then can develop symptoms of congestive heart failure. The heart cannot work effectively if the ventricular rate is too rapid.

If the patient does exhibit any adverse symptoms which suggests possible danger, another treatment may be used; cardio version. Cardio version is also used if digitalization fails to convert the patient to a normal sinus rhythm within a safe period of time. This cardio version uses 20 to 50 watt-seconds.

3. **Atrial Fibrillation**

This arrhythmia occurs due to multiple electrical impulses leaving the atrium. Arteriosclerosis and other diseases can cause scarring of the atrium. This scar tissue becomes "irritable" and begins to send out many impulses across the atria. The ECG tracing will not show a distinct P wave since the rate is extremely rapid (350-600). The P waves appear as an atrial wave or in other words, wavering lines with no distinct P wave visible (see figure below). The patient's pulse is irregular. Since the P waves come so rapidly and at irregular intervals, the ventricular response is irregular and so is the pulse usually.

The patient may or may not have clinical symptoms. Some patients have weakness and hypotension, other patients have no symptoms. The ventricular response usually determines if the patient will by symptomatic or not. If the AV node blocks large numbers of the P waves, the ventricles will then maintain a fairly normal pulse. If the AV node allows many P waves through, the ventricles will respond and the pulse can become very high. As the ventricles respond faster to the P wavers, cardiac output will decrease and the patient may then become symptomatic.



This is A.F. with <u>slow</u> ventricular response. Note the "wavy" appearance of the multiple rapid P waves.

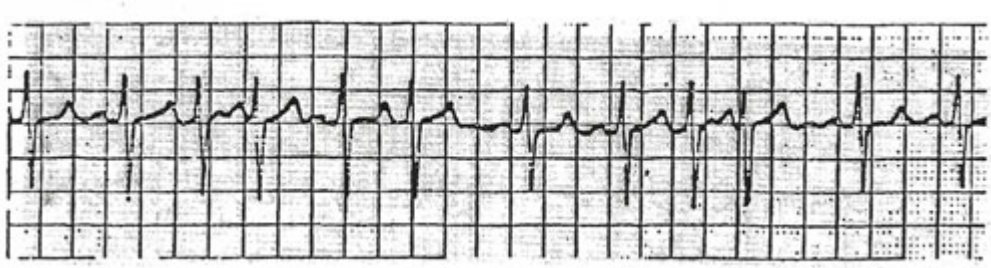
As mentioned, when the patient's pulse rate reaches and passes 100 beats per minute, cardiac output usually begins to drop due to incomplete filling of the ventricles. When the symptoms begin to appear, the patient must be treated, or it may lead to CHF and other complications. If the patient is not already on digitalis, treatment for atrial fibrillation (AF) is digitalization.

This therapy takes several hours, so the patient should be in a stable condition. If digitalis therapy is not practical for some reason, cardio version may be necessary. Cardio version does have risk involved, especially if the patient is already on digitalis. One hundred watt-seconds is usually used for this purpose.

Other treatment methods for AF include the use of quinidine and propranolol. There are also some new drugs which will be available soon to treat this arrhythmia.

4. **Premature Atrial Contractions (PAC's)**

PAC's are ectopic discharges from the atrium causing contraction of the atrium. However, there is not always ventricular contraction following these discharges. The focus of the discharge may be either the right or left atrium. The QRS is almost always normal, but may be aberrantly conducted. Rare PAC's occur normally in most people, but frequent ones may be indicative of organic heart disease.

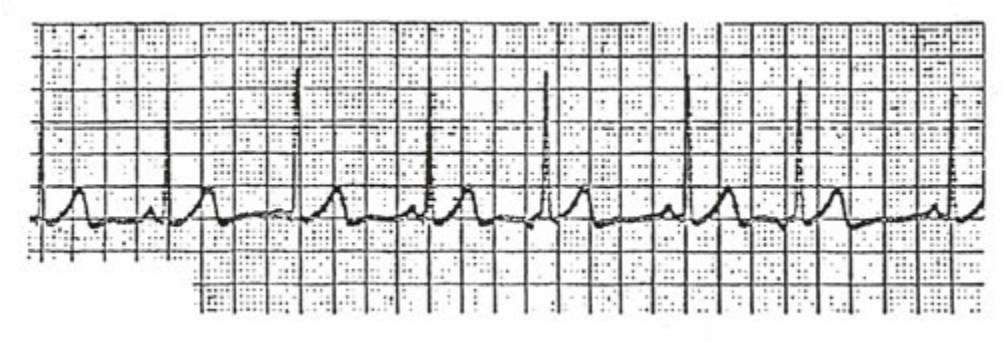


The treatment of PAC's depends upon the frequency of occurrence and the clinical symptoms involved. Usually mild conditions respond to the omittance of stimulants from the diet or to stopping certain drug therapies for example: caffeine, tobacco, amphetamines, alcohol, etc. Mild sedation may also be used to stop mild episodes. In more severe cases, and in cases where there are severe clinical signs/symptoms, drug therapy may be indicated. Drugs commonly used to stop PAC's include digitalis, quinidine, propranolol and procainamide.

5. **Junctional (NODAL) Contractions**
(Also known as Premature Junctional Contractions, PJC's)

Just as the name implies, the disorder is causes by a premature contraction. The pacemaker site of the PJC is an ectopic pacemaker in the AV junction. The rhythm is irregular when the premature contractions are present. P Waves may or may not be associated with PJC's. If they are present, they are abnormal, varying in size, shape, and direction from the normal P Waves. The P Waves are often inverted when present, or they are buries in the QRS complex so they are not visible.

Occasional PJC's may occur in a healthy person without a specific cause. However, PJC's are most often caused by digitalis toxicity, excessive doses of other cardiac drugs, and by enhanced automaticity of the AV junction. PJC's often resemble PVC's (discussed later). Therefore, they must be identified properly in order to have the correct treatment given.



6. **AV Block**

AV Block refers to several different related arrhythmias. The name itself sounds innocent, but several of the related disorders may be life-threatening. By definition, AV Block refers to a disorder of the AV node. There is scarring or inflammation at the node and impulses are slowed or blocked at that point. If the heart block is total or complete, no impulses get through to the ventricles in order to initiate their contraction. AV Block is described in three "stages" in order of the amount of blockage present. There is first degree block, second degree block, and third degree block. Each degree higher represents more severe blockage.

First Degree AV Block

First Degree AV Block is the least serious condition of the three. It is characterized by a prolonged conduction of the impulses through to the ventricles. The actual pathology may be the area of myocardium directly surrounding the AV node itself, or the problem may be in the node. First degree block also frequently occurs in the presence of an acute inferior wall MI> it may also occur as the result of increased vagal (Parasympathetic) tone or digitalis toxicity.

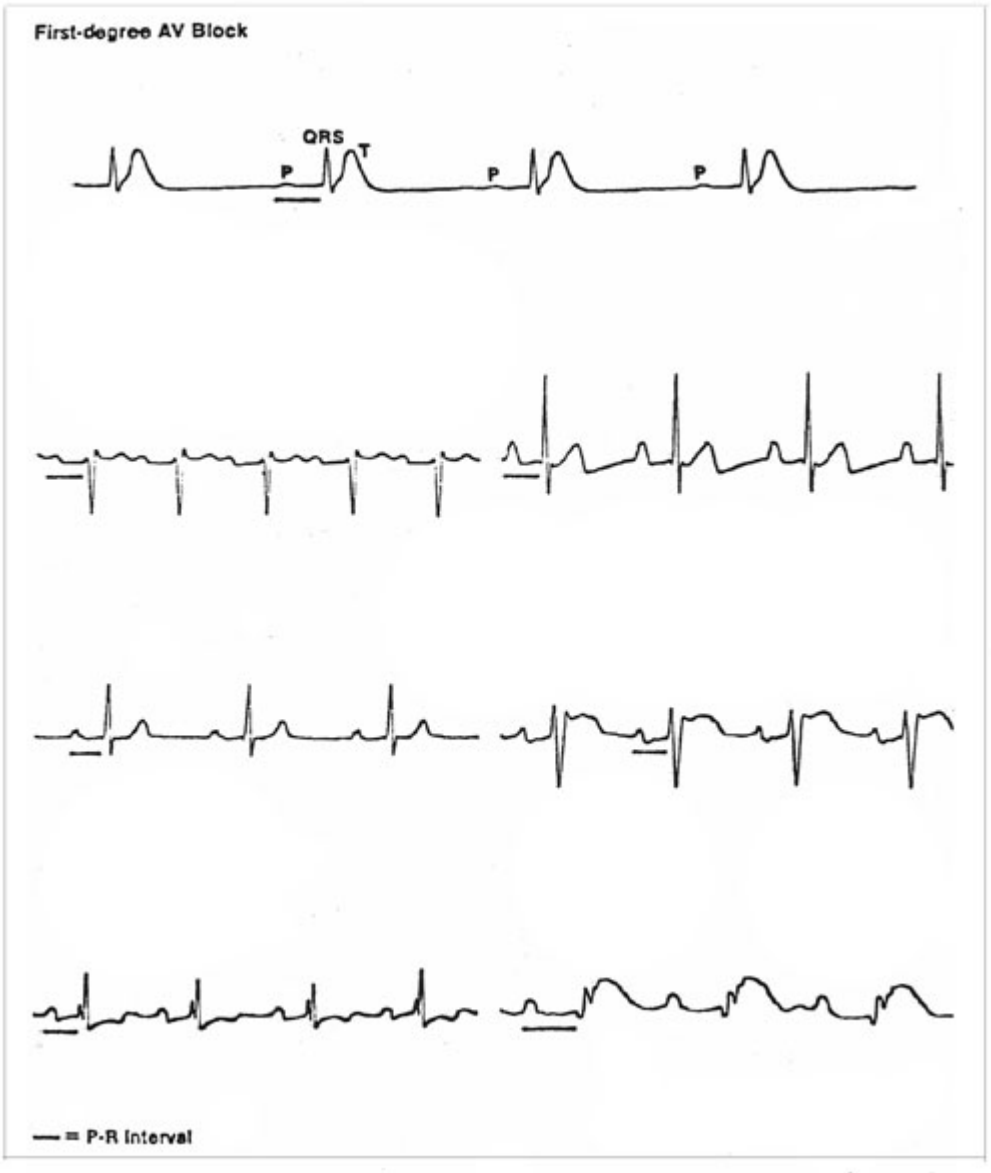
Whichever the problem, the delay in the conduction is apparent on ECG as an increased P-R interval. The PR interval is shown in the figure below. The normal time for the P-R interval is up to 0.20 seconds. If the interval is longer, first degree block is present (assuming no other underlying arrhythmia is present as well).

Also, in the first degree block, every other aspect of the ECG must be normal. The QRS interval is normal. The P-R interval is prolonged but constant. The P waves are identical and precede each QRS complex. Each P wave is conducted to the ventricles. Again, the only abnormality is that the P-R interval is prolonged. The heart rate is that of the underlying atrial or sinus rhythm.

The QRS complexes are usually normal with first degree block. However, they may rarely be abnormal because of a preexisting bundle branch block. Typically, the AV conduction ratio is 1:1, that is, a QRS complex follows each P wave.

First degree AV block usually produces no symptoms in the patient. However, this condition can progress to a higher degree AV block. Because this condition may get worse, patients are usually observed carefully. The person will have regular checkups and regular EKG tracings and monitoring.

See example EKG tracings below:



Second Degree AV Block

This type of arrhythmia is divided into two categories:

The first is called Mobitz Type I or the Wenckebach Phenomenon.

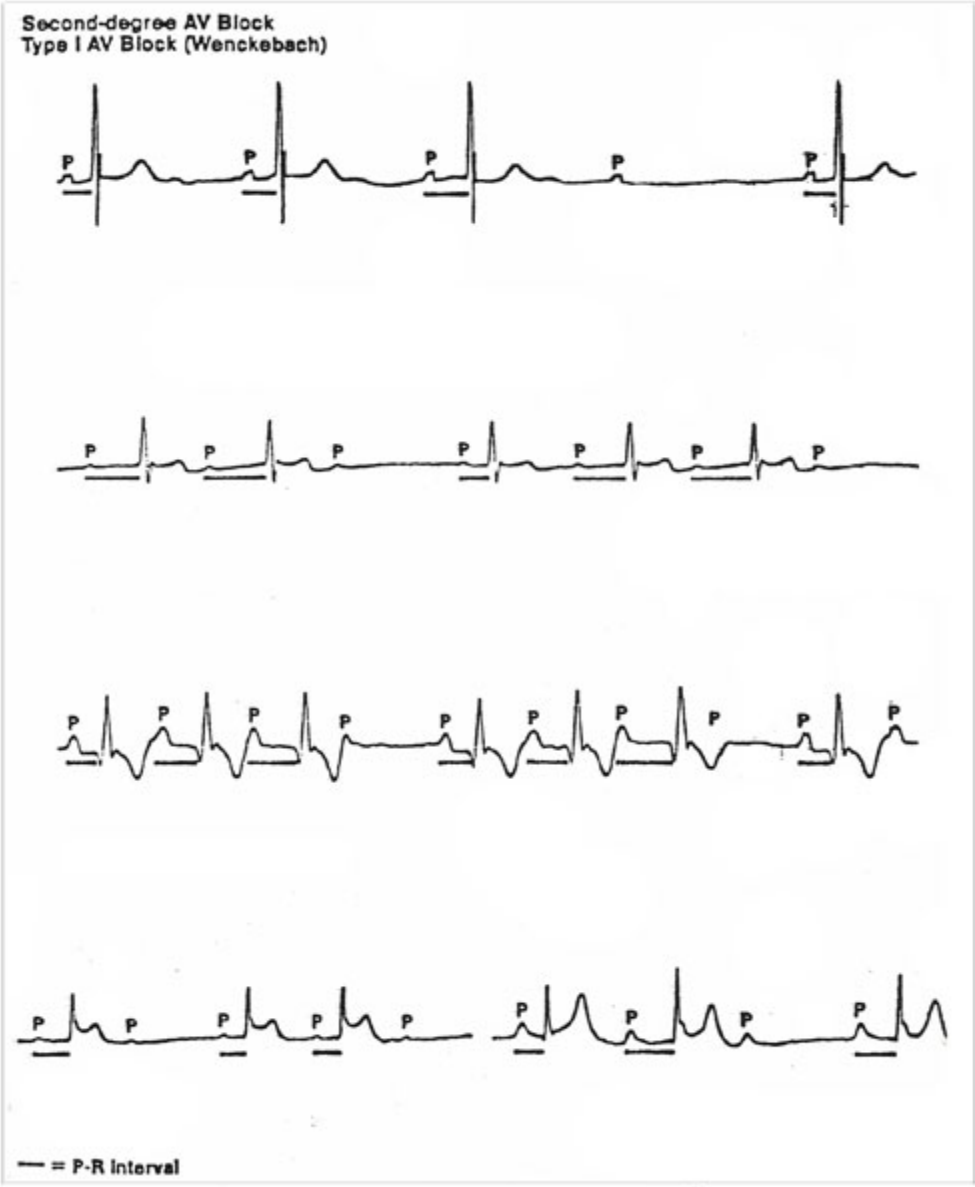
The second is called Mobitz Type II AV Block

In general, both of these are more severe than the previously discussed first degree block. In second degree block, the impairment is so great that some of the P waves fail to initiate a ventricular contraction. In second degree block, the atrial impulses are usually of normal rate and rhythm, and the QRS complexes are also normal. The term Mobitz is being used less and the arrhythmias are simply being called Type I or Type II. However, Mobitz is still used by many authorities.

Mobitz Type I AV Block

In this type of second degree block, there is a progressive increase in the P-R interval as shown in the example. The P-R interval increases until the point that the P wave is totally blocked and no QRS flows, and the beat is dropped. After the dropped beat, the cycle starts over again. The P-R interval appears almost normal, and then continues to lengthen and the cycle repeats itself. This type of second degree block is common in patients with an acute inferior myocardial infarct, but it is generally considered the less serious type of second degree block. It may disappear spontaneously. It may even be caused by excessive digitalis dosing, and usually does not require a pacemaker. However, if the ventricular response is very low, then atropine or isoproterenol may be used. This type of arrhythmia is not serious by itself, but may increase to a more serious arrhythmia. The patient needs to be observed and have regular checkups.

In this type of arrhythmia, the atrial rhythm is essentially regular. The ventricular rhythm is irregular. The P waves are identical and precede the QRS complexes when they occur. The P-R intervals gradually lengthen until a QRS complex fails to appear after a P wave (Non-conducted P wave or Dropped Beat). Following the pause caused by the dropped beat, the sequence begins again.



Mobtiz Type II AV Block

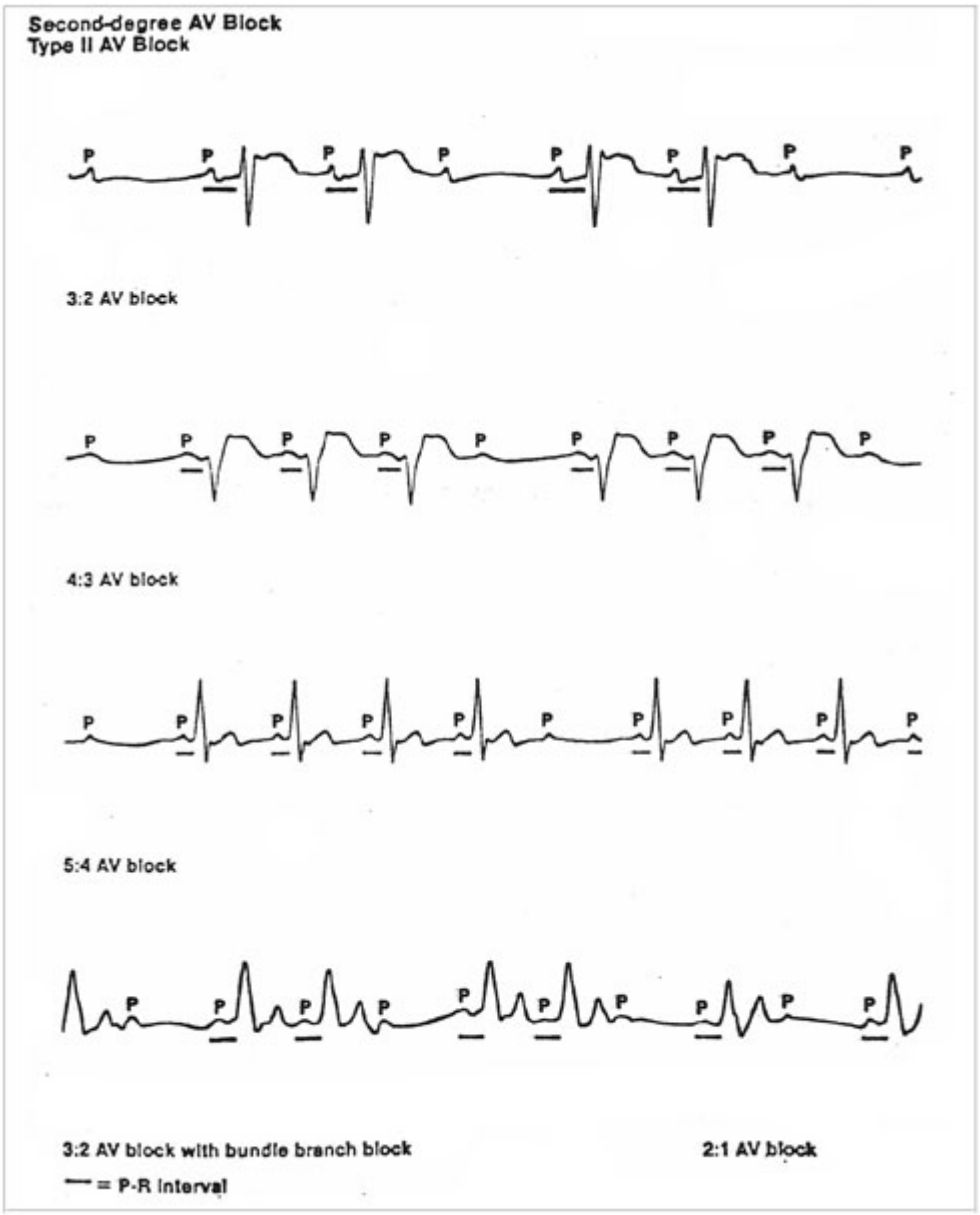
This type of second degree block is the more serious type. Essentially, there is a definite ratio of blocked beats. Some P waves will not get through the AV node in order to initiate the contraction of the ventricles. It can be every 5th beat which is dropped, very 4th beat, or every 3rd beat, etc.

The more frequent the dropped beat, the more serious is the block. If the ratio is 4 to 1, the patient may have not experienced any adverse symptoms. If the blocked beat is every other beat (2:1 ratio) then the person probably will exhibit dizziness, fainting or other symptoms and need immediate treatment. The more serious block, 2:1 ratio, requires immediate treatment because this may lead to ventricular asystole and death.

In this type of block, the atrial rhythm is essentially regular. The ventricular rhythm is usually irregular. The P waves are identical and precede the QRS complexes. The P-R intervals may be normal or abnormal (greater than 0.20 seconds). They are usually constant.

This type of block usually occurs below the bundle of HIS. This represents an intermittent block of conduction of the electrical impulse through one bundle branch and a compete block in the other. This produces an intermittent AV block with an abnormally wide and bizarre QRS complex. Commonly, Type II Second Degree Block is the result of extensive damage to the bundle branches following an anterior-wall myocardial infarction. Rarely, this type of AV block occurs at the level of the Bundle of HIS. When this occurs, the QRS complexes are normal (0.10 second or less) unless a preexisting bundle branch block is present.

This type of block is more serious than type I AV block. The heart rate can be slow enough to cause severe Bradycardia-related symptoms in the patient. This type often progresses to third degree AV block and even to ventricular asystole. The treatment is to immediately insert an external or transvenous cardiac pacemaker. Atropine is usually ineffective in reversing a Type II AV block.



Third Degree Block (Complete AV Block)

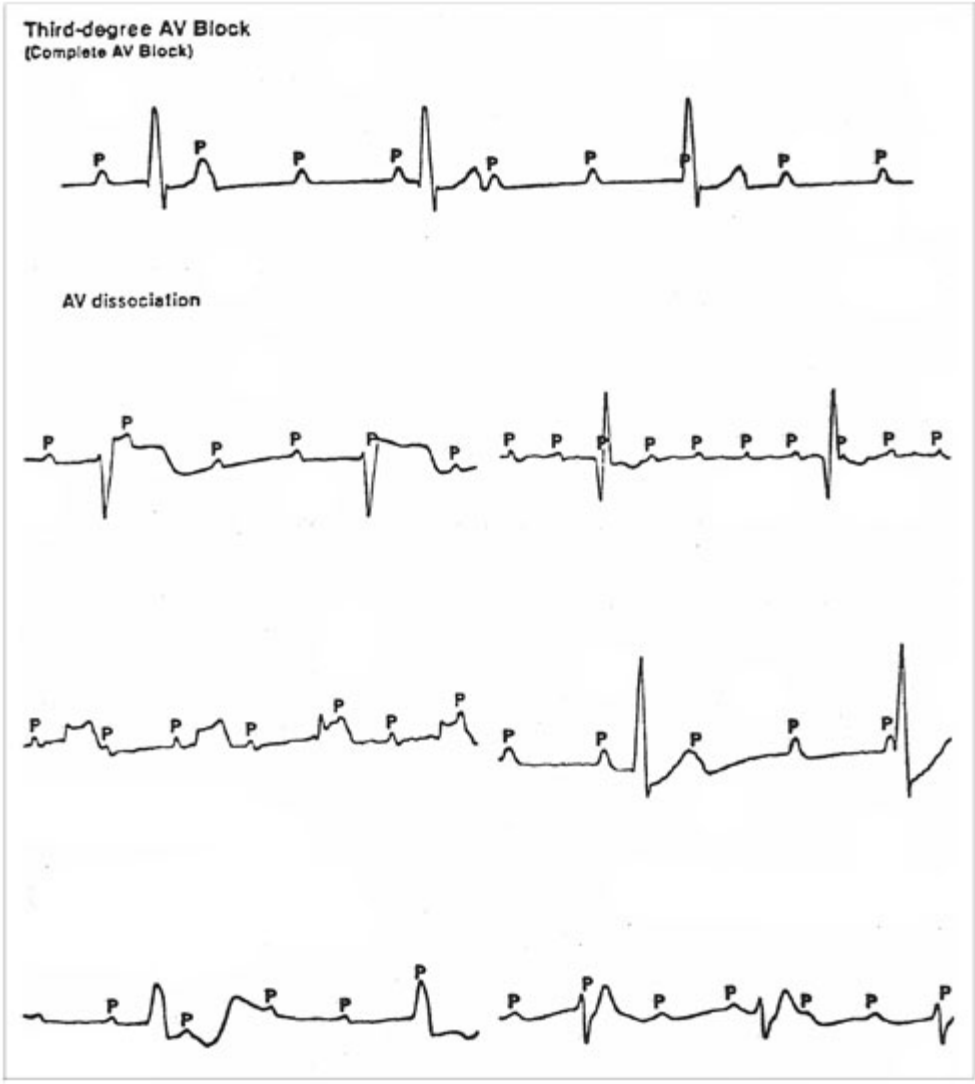
Third degree block is complete or total blockage of all impulses through AV node. The atria and ventricles beat totally independent of each other. The atrial rate and rhythm are usually regular. If P waves are present, they may have originated in the SA node of from an ectopic pacemaker in the atria. P wavers, atrial flutter, or atrial fibrillation waves may be present. When present, they have no relation to the QRS complexes, appearing independently at a rate different from that of the QRS complexes (atrioventricular (AV) dissociation). See example below.

The pacemaker for the ventricles may be anywhere in the conduction system of the ventricles. It may be in the AV node, Bundle of HIS, or bundle of Purkinje system. The lower in the

conduction system that the pacemaker is located, the slower the ventricular rate will usually be. This slow rate is referred to s idioventricular rhythm. If the pacemaker for the ventricle is high in the system, or even the AV node itself, the ventricular rhythm may be sufficient enough that the patient will have no adverse effects. However, since the rate is usually below 60 BPM, the patient usually exhibits Stokes-Admas attacks (syncopal attacks), CHF or angina attacks.

QRS complexes are usually normal, but the QRS interval may be widened and/or lengthened in some cases. The QRS complexes typically exceed 0.12 second and are bizarre if the escape pacemaker site is in the ventricles or if the escape pacemaker site is in the AV junction and a pre-existing bundle branch block is present. However, the QRS complexes may be normal (0.10 second or less), if the pacemaker site is above the bundle branches in the AV junction and no bundle branch bock is present.

Third degree AV block may be transient and reversible. Some treatable conditions may cause this block and it may be reversed. However, when the QRS complexes are wide and bizarre and the heart rate is in the 30 – 40 range, it is an indication that the condition is not reversible. These permanent block conditions are usually cause by the anterior wall MI or chronic degenerative changes in the bundle branches in the elderly. The treatment of third degree block is the immediate insertion of a cardiac pacemaker, no matter what the cause of the block.



7. **Bundle Branch Block**

Definition:

A delayed ventricular stimulation, usually due to blockage of impulses traveling through the bundle of HIS.

Etiology:

This is a conduction defect in either the right or left branches of the bundle of HIS. Usually, the cause is tissue damage.

Clinical Implications:

The arrhythmia itself is not dangerous. However, the cause of the damage is usually the reason for concern. Usually there are no symptoms with bundle branch block (BBB). The problem, as

with other asymptomatic conditions, is usually detected upon routine physical exams, or while the MD is examining the patient for a different complaint.

Right BBB may exist with no symptoms at all and virtually no evidence of disease present. Left BBB is almost always associated with extensive tissue damage (infarcts) of the septal area.

There is no specific treatment for BBB since it does not usually produce any demonstrable symptoms. The nursing implications of BBB are related to nursing observations. Observer for a sudden development of BBB**,** as this should be reported to the MD immediately. In rare cases, BBB may develop as a result of the patient being on digitalis or certain other anti arrhythmic drugs. If this reason is suspected, report to the MD before administering any further doses of the drug.

EKG Findings	
Rate:	Usually normal.
P waves:	Normal.
QRS:	1. Wider than normal (0.12 seconds or greater). 2. The configuration is distorted.
T waves:	Directed opposite the QRS complex.

Dynamics:

The ventricles are contracting asynchronously. This sometimes causes an "M" or "W" shaped QRS complex. Even though they fire asynchronously, there is still little interference with the pump action of the heart, and virtually no functional interference with the heart action.

Branches of the Bundle of HIS

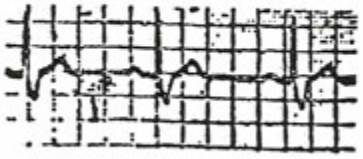
1. **Right Bundle Branch**
2. **Left Bundle Branch (2 Divisions)**
    a. **Anterior-superior division**
    b. **Posterior-inferior division**

Bundle Branch Block, as stated, is abnormal conduction through the ventricles. If a block exists in one of the bundle branches of the Bundle of HIS, the impulse will travel down the branch to the opposite bundle first. Having activated this bundle, the impulse will then spread through the septum to the ventricle on the other side of the block and then cause it to be activated. The block may only affect one division on the left bundle branch.

Left BBB

Right BBB

Left BBB



Right BBB

Wandering Atrial Pacemaker

This arrhythmia may be a normal phenomenon or it may be a warning of a possible atrial arrhythmia. On EKG it is noted that the pacemaker of the heart shifts from the normal SA node to the Atria or to the AV Junction. This may be a normal phenomenon seen in the very young, athletes, or in the aged. It is caused in the majority of cases by the inhibitory vagal (parasympathetic) effect of respiration on the SA node or the AV Junction. It may also be caused by the administration of digitalis of in the digitalis toxicity.

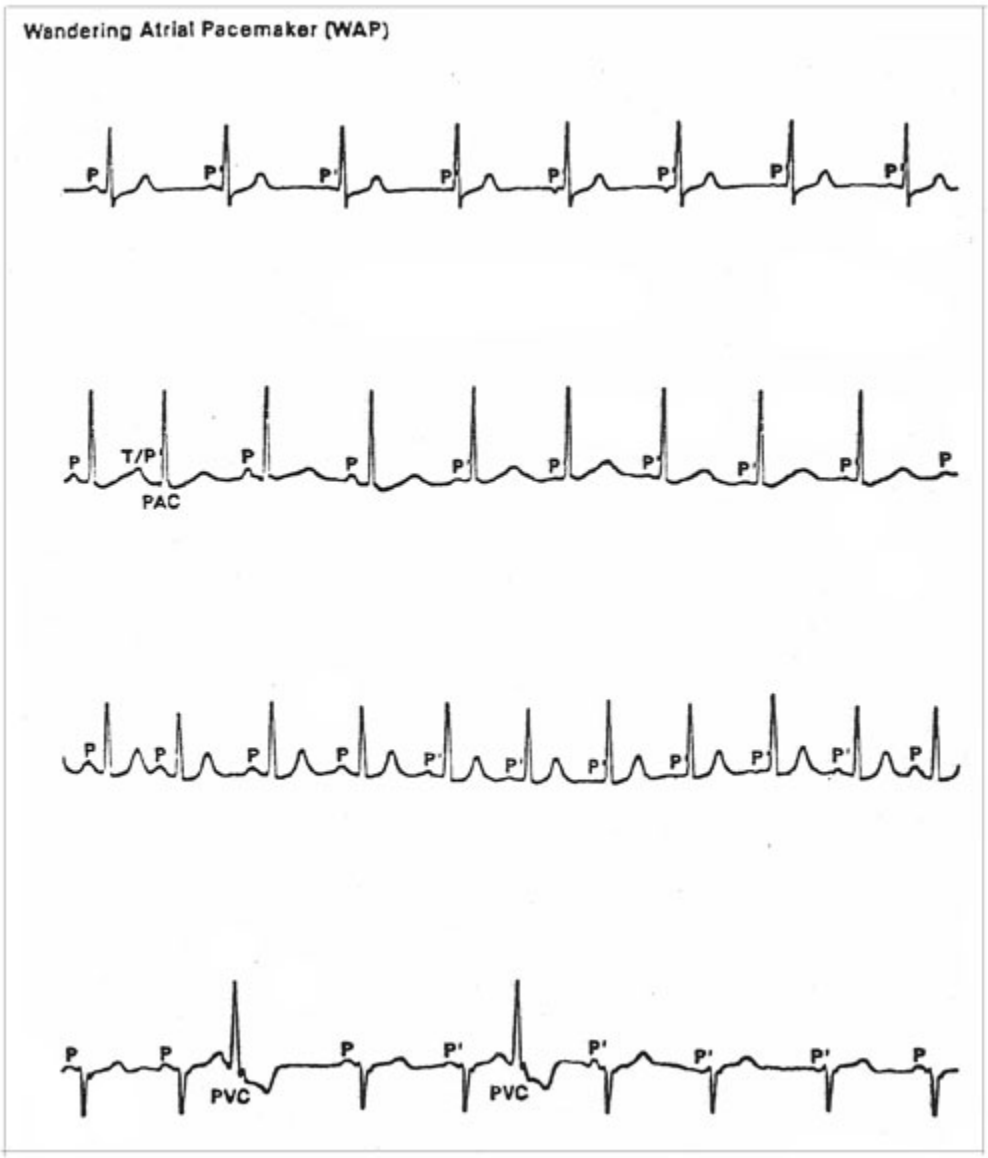
The P wave will precede each QRS complex. However, they will vary in size and configuration, as the pacemaker shifts back and forth from the SA node to AV junction. The P wave may even become inverted (negative) and it may even become buried in the QRS complex. The P waves, other than those arising from the SA node, are called ectopic P waves, or P waves (P prime waves).

The P-R interval will vary along with the location of the pacemaker site. It will usually be about 0.20 second when from the SA node, and shortens to about 0.10 second when the pacemaker is in the atria or the AV Junction. The duration of the intervals reverses as the pacemaker site shifts back to the SA node.

The R-R intervals are usually unequal, but they occasionally may be equal. They increase in duration as the pacemaker site shifts from the AS node to the atria or the AV Junction. The R-R intervals decrease again as the pacemaker site shifts back to the SA node.

The patient's heart rate is usually 60 to 100 beats per minute, but may be slower. Usually, the heart rate gradually slows slightly when the pacemaker site shifts from the SA node to the atria or the AV Junction. It increases as the pacemaker site shifts back to the SA node. The heart rhythm usually irregular, but can occasionally be regular.

A wandering atrial pacemaker is usually not clinically significant. Treatment is not usually indicated. When the heart rate slows excessively, the signs and symptoms, clinical significance, and management are the same as those in symptomatic or marked Sinus Bradycardia.



Ventricular Arrhythmias

1. Premature Ventricular Contractions (PVC's)

PVC's are extra beats which occur from an ectopic focus on the ventricle wall. This focus is usually below the bifurcation of the bundle of HIS. In the normal person these may be caused by smoking, alcohol, or coffee ingestion. They usually are rare and inconsequential in normal persons. PVC's may also, and more frequently, occur as the result of an MI or due to arteriosclerotic heart disease. This irritable spot on the myocardium sends out a powerful

electrical impulse which spreads across the ventricles, causing them to contract out of proper sequence. In other words, the ventricles contract before they have had a chance to completely fill with blood from the contraction of the atria.

PVC's may be unifocal (from one spot on the ventricle wall) or they may be multifocal (from two or more different spots [foci] on the ventricle wall). Obviously, the multifocal PVC is the more dangerous condition; it indicates the general irritability of the myocardium and the possibility of even more dangerous heart arrhythmias. Shown in the below illustration are different types of PVC's.

PVC's may be single and isolated (rare), which are usually normal. They may also be more frequent, occurring at regular intervals.

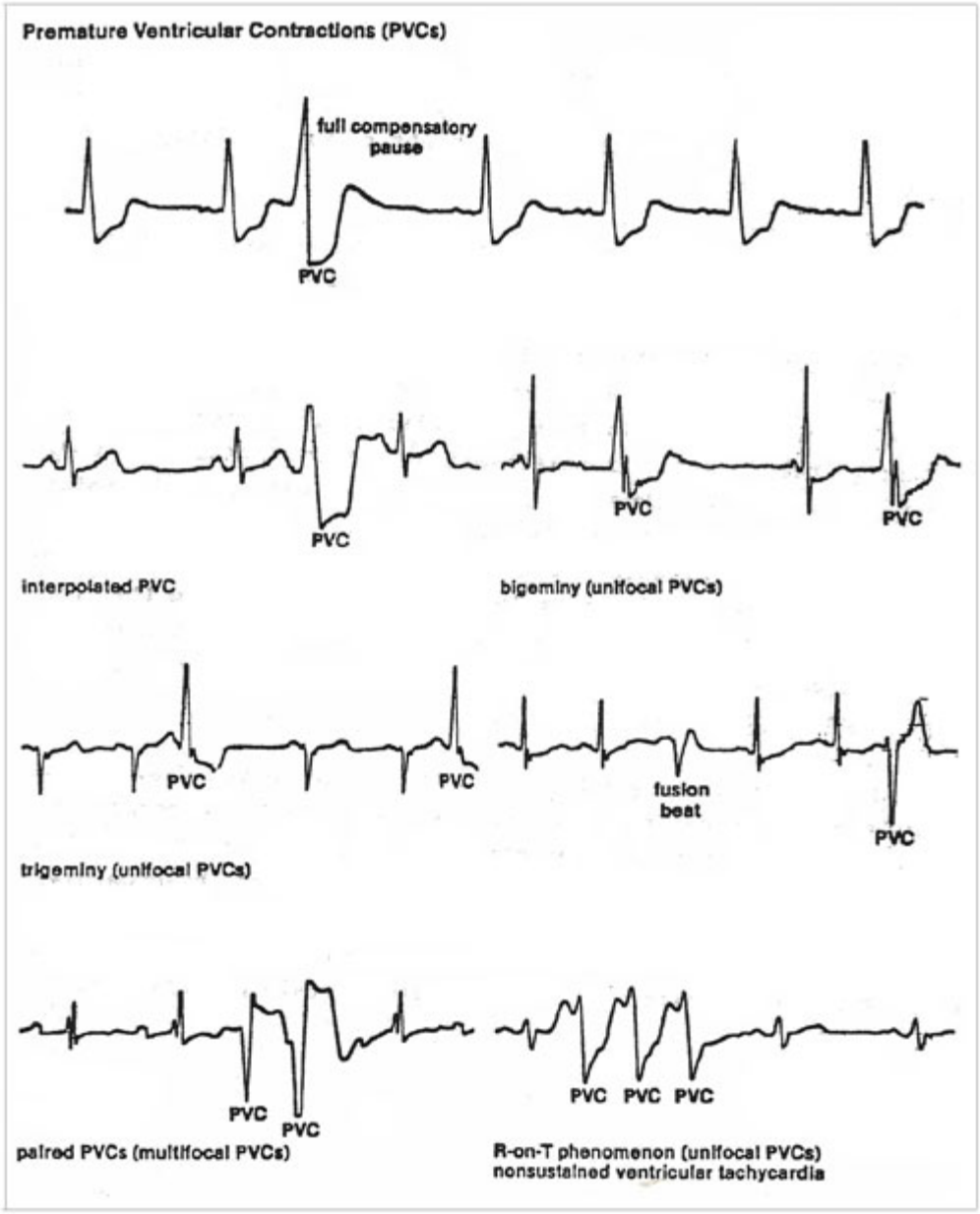
When they occur at regular intervals, they are called:

Bigeminy – every other beat.
Trigeminy – every third beat.
Quadrageminy – every fourth beat

If the beats occur less frequently than every fourth beat there is usually no regular pattern. They will tend to be irregular in the pattern. However, it can be noted that PVC's might occur regularly, every fifth beat, sixth beat, etc.

If they occur more frequently than every 4^{th} beat the condition can be serious and possibly require treatment. Multifocal PVC's are also more dangerous then unifocal. If they appear in groups of two or more together (coupled), the situation could also be dangerous. In addition, the most dangerous situation is called the R-on-T Phenomenon. When the PVC falls on a T wave from the previous contraction, ventricular fibrillation and death can occur. During the T wave (repolarization), heart muscle is very sensitive to outside stimulus thus a strong PVC can send the myocardium into fibrillation. Treatment of PVC's is complex. In the "normal" situation, Lidocaine is administered to decrease the irritability of the myocardium. An initially high intravenous dose (bolus) is given and then the patient is monitored on a lower maintenance dose (intravenous drip). If the PVC's continue after the Lidocaine is terminated, the patient is maintained on an oral dose of some similar type drug.

A case where Lidocaine may not be used would be in Bradycardia. If the SA node rate falls below 60 per minute, the heart may try to compensate by the use of PVC's. Another case is after carefully studying the ECG tracing and determining that the underlying arrhythmia is Sinus Bradycardia, Lidocaine may not be used. The MD will usually give Atropine to increase the SA node firing. This increase in the pulse rate will then give rise to the termination of the inefficient PVC's needed to maintain circulation.



2. Ventricular Tachycardia (V-Tach)

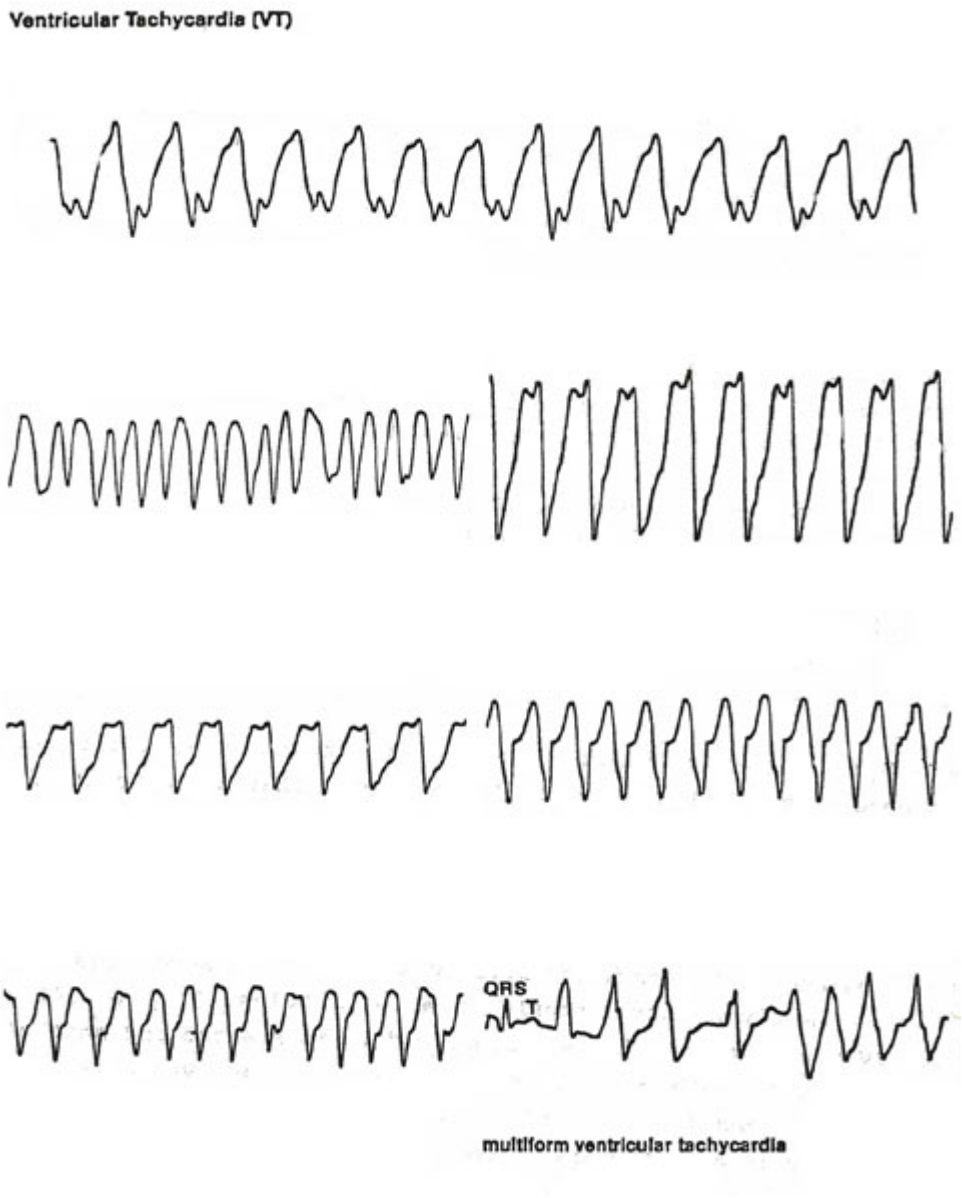
This is a very serious arrhythmia. Whenever three or more consecutive PVS's are seen, at a rate of 100 bpm or more, the term used is Ventricular Tachycardia (V. Tach). In the strictest definition, V. Tach is the same as PVC's, except that there are many of them in a row. The onset and termination of V. Tach may be may be abrupt or not. V. Tach may occur in paroxysms of three or more PVC's separated by the underlying rhythm (non-sustained V Tach or Paroxysmal Ventricular Tachycardia), or persist for a long period of time (sustained ventricular tachycardia). The rhythm is usually regular, but it may be slightly irregular.

When V. Tach occurs, the ventricles do not have sufficient time to fill and thus, cardiac output is greatly reduced. This arrhythmia may also lead to ventricular fibrillation and death. The pacemaker site for V. Tach is an ectopic pacemaker in the bundle branches, Purkinje network, or in the ventricular myocardium itself.

The rate of V. Tach is from about 100-250 bpm. P Waves may be present or absent. P Waves are usually not seen if the rate is increased. If present, the P Waves have no relation to the QRS complexes of the V. Tach. P Waves, if present, may be positive or negative in Lead II. If P Waves are present and occur independently of the QRS complexes, the P-R intervals will vary widely.

QRS complexes are described as "wild-looking" and with great swings and exceed 0.12 second. They are followed by large T Waves that are opposite in direction of the major deflection of the QRS complexes. The QRS complexes may look alike in shape and form or they may be multiform (markedly different from beat to beat).

The treatment of V. Tach is essentially the same as for severe cases of PVC's. Lidocaine is given intravenously in a large bolus, 75mg to 100mg over two minutes IV push. Meanwhile, a Liodocaine drip is started and another bolus is given if the V. Tach does not stop. Two new drugs, Verapamil and Bretylium may also be used for this and other similar arrhythmias. If the drug therapy fails, or if drug therapy is contraindicated, cardio version may be used. Quick treatment is necessary, as death can result quickly.



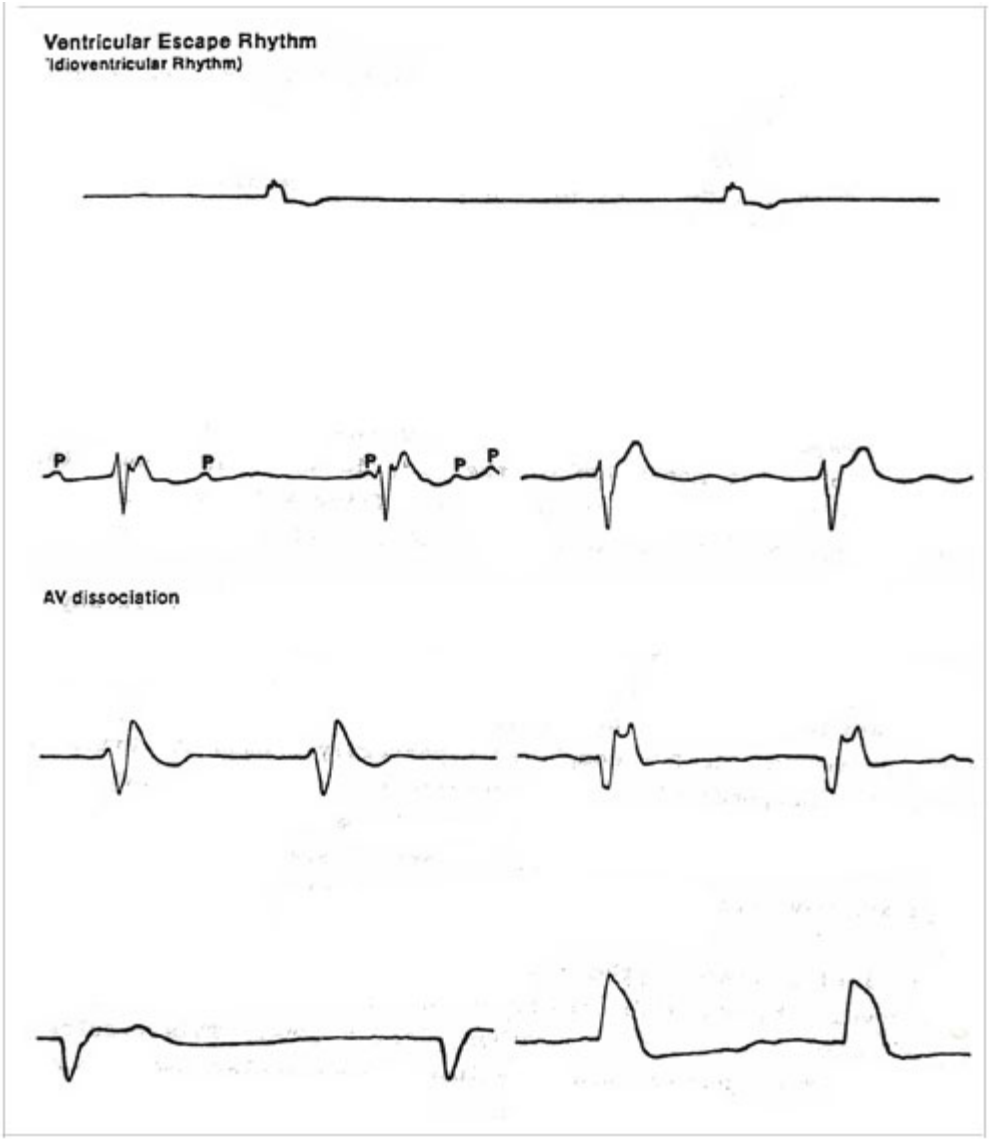
3. Ventricular Fibrillation

Ventricular Fibrillation (V. Fib) means sudden death. The blood pressure drops immediately to zero and so does the cardiac output. The heart is merely quivering due to the rapid multiple electrical discharges in the myocardium. V. Fib is one of the most common causes of cardiac arrest. It usually occurs in the presence of significant cardiac disease. It occurs most commonly in coronary artery disease, myocardial ischemia, acute myocardial infarction, and third degree AV Block with a slow ventricular response. V. Fib may also occur in cardiomyopathy, mitral valve prolapsed, cardiac trauma (blunt trauma), and in digitalis toxicity. V. Fib may also occur during anesthesia, cardiac and noncardiac surgery, cardiac catheterization, during cardiac pacing, following cardio version, accidental, or non-accidental electrocution.

A PVC may also initiate V. Fib when the PVC occurs during the vulnerable period of ventricular repolarization, coincident with the peak of the T. Wave (i.e. R on T phenomenon), particularly when electoral instability of the heart has been altered by ischemia and acute MI. Sustained V. Tack and Ventricular flutter may precipitate V. fib.

The impulses are discharging from many random foci and the heard cannot respond with an organized contraction. There is no specific pattern to the discharge. No QRS complexes can be seen, no P waves are present, no P-R intervals, and no R-R intervals can be seen. The ECG shows different types of wavering baseline patterns in the presence of V. Fib. Shown in the below figure are several examples of fibrillation, course fibrillation, fine fibrillation, and more. Course fibrillation is more likely to be reversed because it indicates a recent onset of fibrillation. Fine fibrillation indicates a more advanced fibrillation and is less likely to be reversed with treatment.

The treatment for this condition is defibrillation by DC shock. CPR will be started until the defibrillation can be performed. The large electrical shock to the myocardium stops the fibrillation and allows the heart to return to its normal rhythm.



Ventricular Escape Rhythm (Idoventricular Rhythm)

This rhythm is characterized by a heart rate usually between 30 to 40 bpm. but may be lower than 30. An escape rhythm refers to the "automatic" or "escape" pacemaker of the heart located in the bundle branches, Perkinje network, or ventricular myocardium. When the "normal" pacemaker (usually the SA node) is blocked, the escape mechanism takes over. This can be caused by sinus arrest, third degree heart block, and other heart problems that block the normal pacemaker.

Ventricular escape rhythm is a "protective" mechanism of the heart. It allows the heart to keep beating (even though is it at a very slow rate) when there is a major blockage of the impulses that make the ventricles beat. Another protective mechanism of the body is to "faint" when this happens. More blood goes to the brain when you faint and lay flat on the floor and the ventricles continue to beat at the idoventricular rate.

The ventricular escape rhythm is usually regular but it may be irregular. P Waves may be present or absent. If present, they do not have a set relation to the QRS complexes. If present, the P Waves may be positive (upright) or negative (inverted). If present, P Waves may precede, be buried in, or follow the QRS complexes haphazardly. When the atria and ventricles beat independently, **atriventricular (AV) dissociation** is present. P-R intervals are absent. R-R intervals may be equal or may vary. QRS complexes exceed 0.12 second and are bizarre. Sometimes the shape of the QRS complexes may even vary in each different lead.

Ventricular escape rhythm is usually very symptomatic. The patients will usually develop hypotension with marked decrease in cardiac output and decreased perfusion of the brain and other vital organs. This results in syncope, shock, and congestive heart failure. Ventricular escape rhythm must be treated promptly in order to reverse the consequences of the reduced Cardiac output.

Chapter III Arrhythmia Determination

This is one of the most important sections of this course. Interpretation of the ECG must be done in a logical sequence in order to best serve the patient. There are many different approaches to the interpretation of the ECG, but in this section we will present a "common sense" approach. If you are responsible for a patient who is being monitored, you want to be able to recognize any arrhythmia that may be life-threatening. You want to be able to do that as fast as possible; it may save the life of your patient. As you read and study these steps, remember that they may not apply to every patient in every situation. However, you should always be deliberate and methodical when you interpret the ECG.

Phase I: Assessment

a. Assess patient symptoms, if and, and vital signs.
b. Assess leads to the patient (leads must be in proper placement).
c. Assess obvious abnormalities of ECG (rate, rhythm).

In the assessment phase, the nurse must quickly note any symptoms. The symptoms will determine if the arrhythmia is severe or not. The vital signs are important. At the same time you are assessing the patient, look at the ECG to determine if there are any gross arrhythmias. If so, you can then take the appropriate action. The patient may need immediate resuscitation or they may be able to wait for treatment.

Phase II: ECG Components (Examine the Individual Components of the EKG)

a. Step One: Identity the QRS complex.
b. Step Two: Determine the heart rate.
c. Step Three: Determine the ventricular rhythm.
d. Step Four: Identify the P. Waves.
e. Step Five: Determine the P-R or R-P interval.
f. Step Six: Determine the pacemaker rate.

Phase III: Determine the Arrhythmia

a. Atrial arrhythmia.
b. Ventricular arrhythmia.

Once you have identified the abnormal component of the ECG, you then name the arrhythmia. If the abnormality is in the atria (P wave), then identify the arrhythmia. If the abnormality is in the ventricle, then identify the arrhythmia.

Phase IV: Action

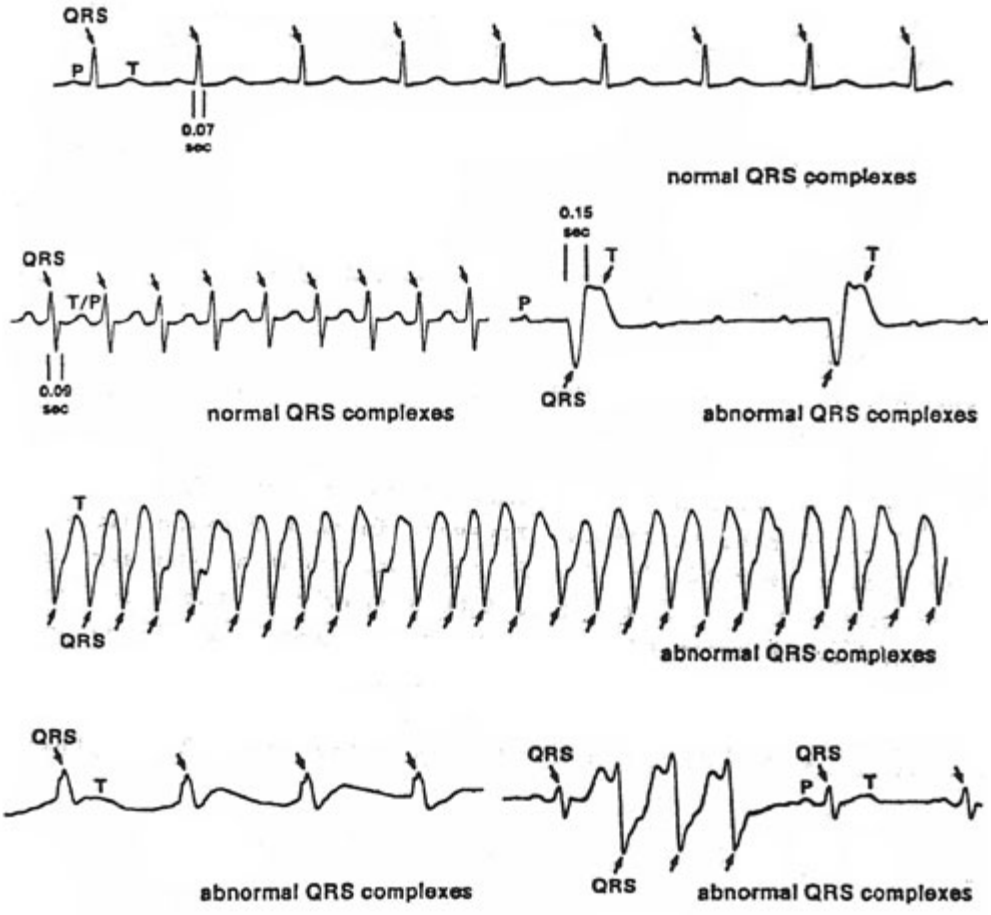
a. Immediate action.
b. Long-term action/intervention

As stated above, if the arrhythmia is immediately life-threatening, then immediate action must be taken. However, in most nursing situations the action will involve notifying the MD and then treating the arrhythmia with the appropriate drug. As you proceed through each of the steps, you must continually be aware of the changes in the patient's condition and of possible intervention. Each hospital has a different protocol for dealing with arrhythmias. Always consider your hospital's policy and procedure and use your common sense when dealing with these potentially fatal arrhythmias.

Step One: Identify the QRS Complex

The QRS complexes, consisting of one or more positive and negative deflections called Q, R, and S waves are identified.

The duration and shape of the QRS complexes are noted. The QRS complexes may be normal (0.10 second or less wide) or abnormal (greater than 0.10 second wide and bizarre appearing). The QRS complexes of ventricular arrhythmias are typically wide and bizarre. The QRS complexes may also be wide and bizarre in supraventricular arrhythmia – one that originates in the SA node or an ectopic pacemaker in the atria or AV junction – if a bundle branch block or aberrant ventricular conduction is present. Less commonly, anomalous AV conduction is the cause of abnormal QRS complexes in arrhythmias originating in the SA node or atria.



Step II: Determine the Heart Rate

The heart rate, as calculated using the ECG paper tracing, is the number of ventricular depolarization's (QRS complexes) or beats occurring in one minute. The heart rate can be determined by using the six-second count method, a heart rate calculator ruler, the R-R interval method, or the triplicate method.

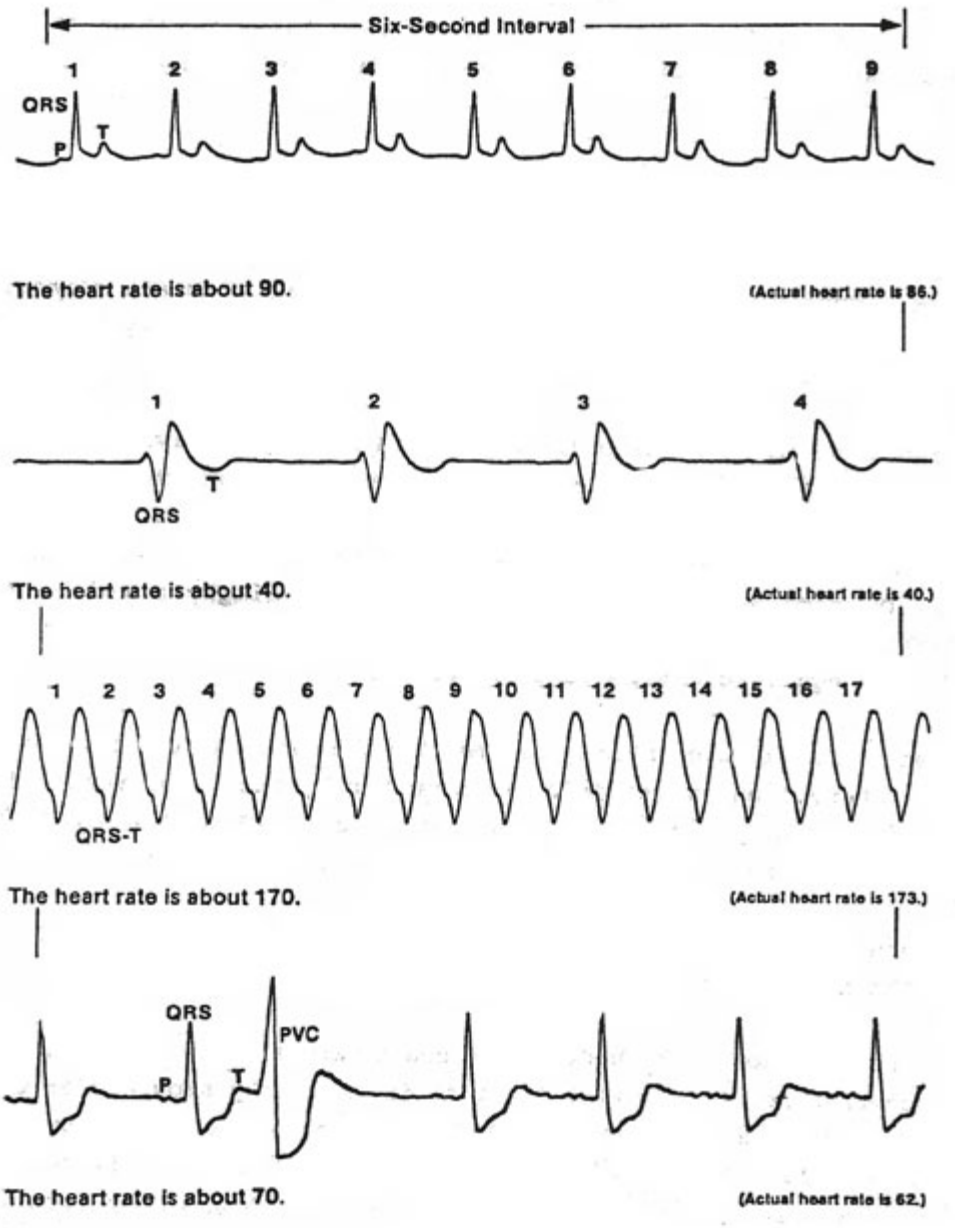
The six-second count method is the simplest way of determining the heart rate. It is generally considered the fastest method, with the possible exception of using the heart rate calculator ruler method. However, the six-second count method is probably the least accurate method. It will however, give you a fairly accurate rate and this method may be used when the heart rhythm is either regular or irregular.

The short, vertical lines at the top of most ECG papers divide the ECG strip into three-second intervals, when the EKG paper is run at the standard speed of 25mm per second. These 3-second lines are at the top of the paper and it may be a separate (separate from the small square area) dark vertical line. On some ECG papers, these markers are simply a thickened part of the line that is already at the top of the small squares. Two of these intervals are equal to a six-second interval.

The heart rate is calculated by determining the number of QRS complexes in a six-second interval and multiplying this number by ten. The result is the heart rate in beats per minute. The heart rate calculated by this method is almost always an approximation of the actual heart rate.

Example: you counted nine QRS complexes in the six-second interval. The heart rate is 9 X 10 = 90 bpm.

This above method works best if the heart rate is very regular. To obtain a more accurate heart rate when the rate is extremely slow and/or the rhythm is grossly irregular, the number of QRS complexes should be counted for a longer interval. An example would be to count for two six-second intervals and then adjust your multiplier. If there are fifteen QRS complexes in a twelve-second interval then 15 X 5 = 75 bpm. This is probably more accurate than just counting for sex seconds. There are other methods of determining heart rate, these will be covered later.



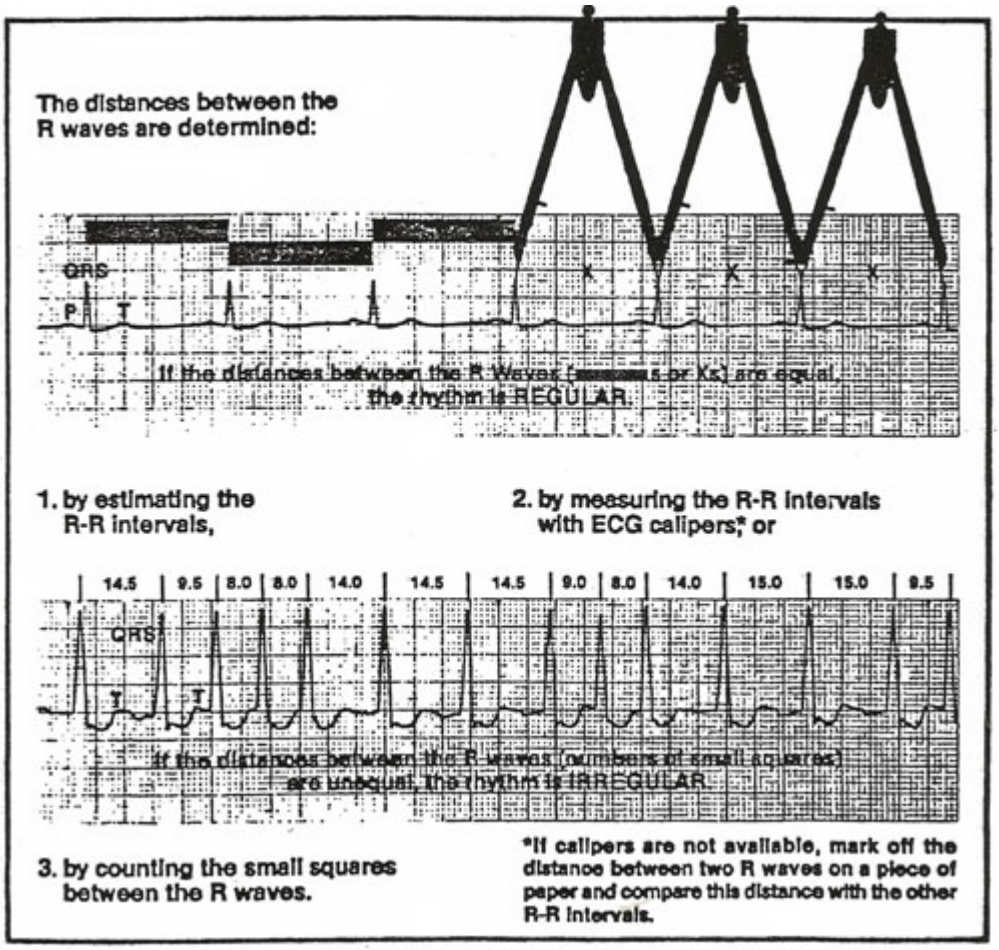
Step III: Determine the Ventricular Rhythm

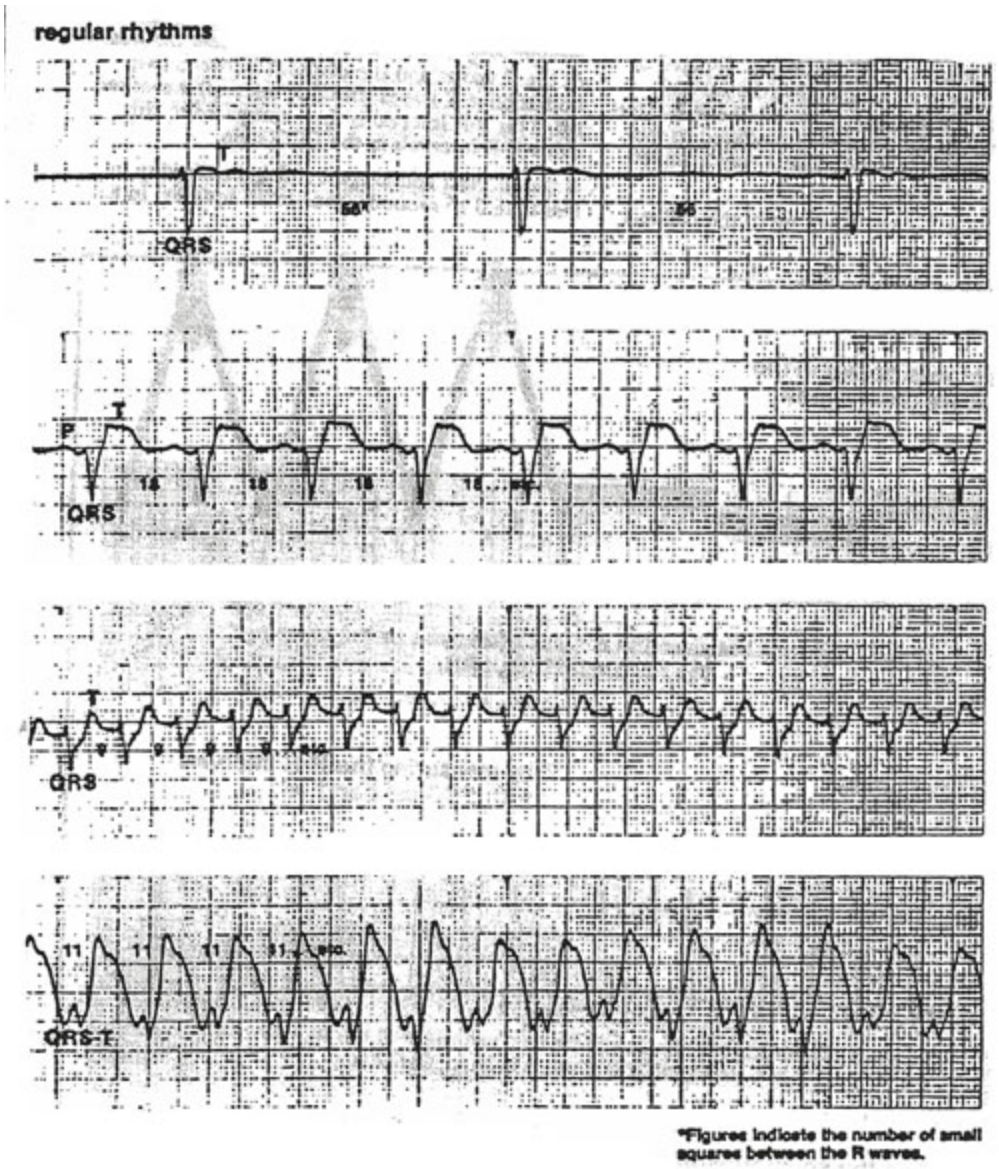
The rhythm is determined by comparing the R-R intervals to each other using ECG calipers or, if calipers are not available, a pencil and paper. First, an R-R interval (preferably one located on the left side of the ECG strip for the sake of convenience) is measured. Second, the R-R intervals in the rest of the strip are compared to the one first measured in a systematic way from left for right.

If ECG calipers are used, one tip of the calipers is placed on the peak of one R wave; the other is adjusted so that it rests on the peak of the adjacent R wave. Without changing the distance

between the tips of the calipers, the other R-R intervals are compared to the R-R interval first measured. If a pencil and paper are used, the straight edge of the paper is placed near the peaks of the R waves and the distance between two consecutive R waves (the R-R interval) is marked off. This R-R interval is then compared to the other R-R intervals in the ECG strip.

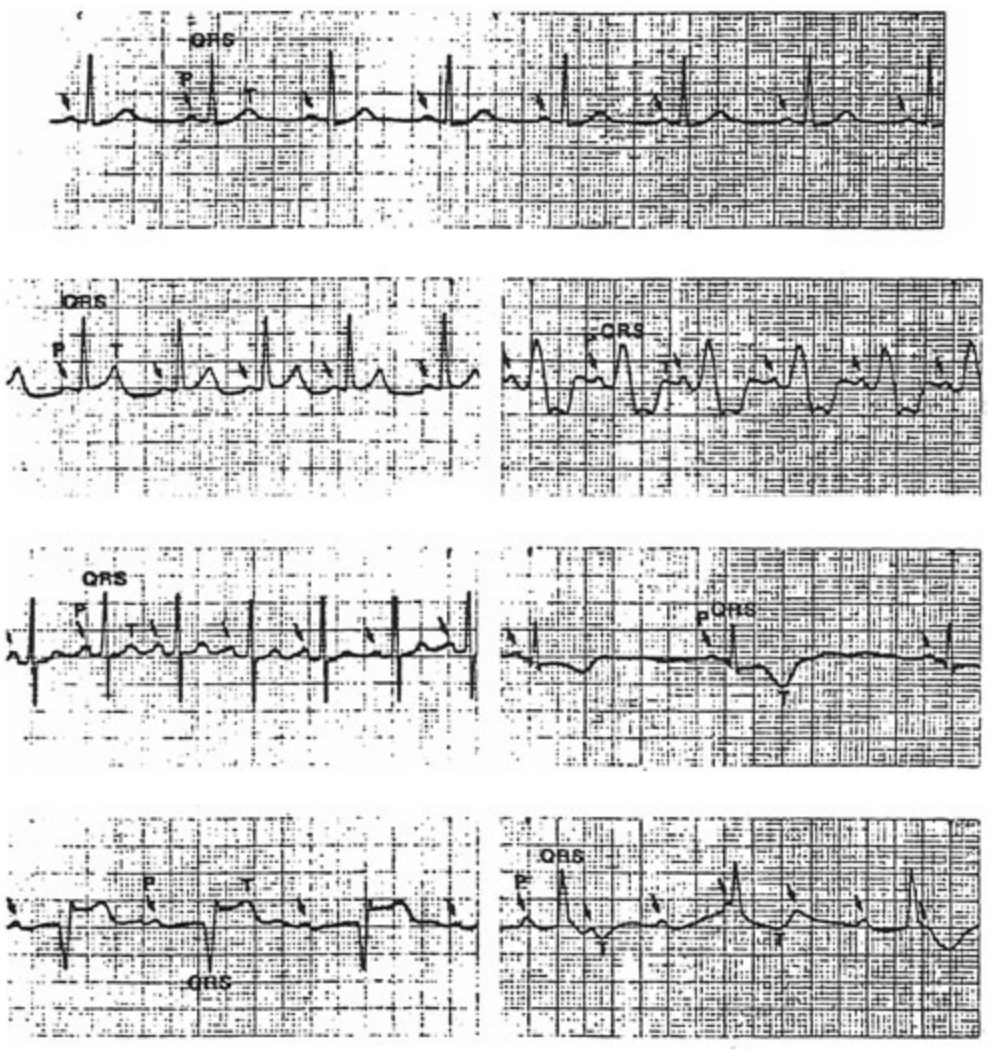
If the shortest and longest R-R intervals vary by less than).16 seconds (four small squares) in a given ECG strip, the rhythm is considered to be "essentially regular." (Thus, the R-R intervals of an "essentially regular" rhythm may be precisely equal or slightly unequal.) If the shortest and longest R-R intervals vary by more than 0.16 seconds, the rhythm is considered to be irregular. The rhythm may be slightly irregular, occasionally irregular, regularly irregular, or irregularly irregular. Other terms to describe an irregularly irregular rhythm are grossly and totally irregular.





Step IV: Identify the P Waves

A normal P wave is a positive, smoothly rounded wave appearing before each QRS complex or slightly without a QRS complex following it. It is 0.5 to 2.5 mm high and 0.10 second or less wide. An abnormal P wave may be positive, negative, or flat (isoelectric).



Step V: Determine the P-R Interval

The P-R interval is determined by measuring the distance in seconds between the onset of the P wave and the onset of the first wave of the QRS complex, be it a Q, R, or S wave. A normal P-R interval is 0.12 to 0.20 second in duration. It also indicates that the electric impulse causing the P wave, originated in the AS node or in an ectopic pacemaker in the upper of middle part of the atria. When the heart rate is fast, the P-R interval is shorter then when the heart rate is slow.

A P-R interval less than 0.12 second or greater then).20 second is abnormal. An abnormally prolonged P-R interval indicates a delay in conduction of the electrical impulse through the AV node or ht bundle of HIS (AV block). A P-R interval less than 0.12 second indicates that the electric impulse has originated in an ectopic pacemaker in the lower part of the atria or in the AV junction or that the eclectic impulse progressed from the atria to the ventricles through antriventricular (AV) conduction pathways other than the AV node and bundle of HIS (anomalous AV conduction).

If a P wave follows the QRS complex, an R-'P interval is present. This indicates that ht electrical impulse responsible for the P' wave and QRS complex has originated in an ectopic pacemaker in the AV junction or ventricles. An R-P' interval is usually 0.20 second or less.

Step VI: Determine the Pacemaker Site of the Arrhythmia

Use the following chart to determine the pacemaker site of Arrhythmias with P waves associated with the QRS complexes:

Pacemaker Site	Direction of P Wave	P/QRS Relationship	P-R Interval
SA Node or Atria	Positive (upright)	P precedes QRS	0.12 -0.20 second or greater OR less than 0.12 second
Lower Atria OR Upper AV Junction	Negative (inverted)	P precedes QRS complex	Less than 0.12 second
Lower AV Junction OR Ventricles	Negative (inverted)	P follows QRS complex	NONE

Use the following chart to determine the Pacemaker site of the QRS complexes **NOT** associated with P waves:

Pacemaker Site	QRS Duration	QRS Appearance
AV Junction	0.10 second or less	Normal
AV Junction* OR Bundle Branch	0.10 – 0.12 second	Normal
AV Junction* OR Purkinje Network, Ventricular Myocardium	Greater than 0.12 second	Bizarre

*In association with a preexisting incomplete bundle branch block or an aberrant ventricular conduction.
**In association with a preexisting complete bundle branch block of an aberrant ventricular conduction.

Chapter IV Arrhythmias and Select Disease Conditions

Cardiovascular Disease and the ECG Possible Arrhythmias

1. Myocardial Infarct	Heart block, PAC's, PVC's, tachycardia
2. Congestive Heart Failure	Tachyarrhythmia's (sinus, tachycardia, ventricular tachycardia) PVC's
3. Cardiogenic Shock	Tachyarrthymias, PAC's, PVC's, cardiac standstill
4. Pericarditis	Tachycardia, PVC's, and ventricular arrhythmias
5. Open Heart Surgery	Heart block, tachyarrhythmia's, other conduction deficits

The above is merely a guide to possible complications with certain cardiac disorder. There can be others as well. If the patient in question is being monitored in the ICU or other critical care unit, any of the arrhythmias will be detected rapidly. If the patient is on the med-surg unit and not being monitored, the nurse MUST be acutely aware of the possibility of arrhythmias. Look for such indications as changes in the level of consciousness, dizziness, pallor, confusion, lowered urinary output, sudden development of edema, or other signs/symptoms which might indicate and arrhythmia. Of course, nothing will take the place of careful and accurate recording of vital signs. A sudden or even insidious change in the vital signs can be the first indication of an arrhythmia.

Fluid and Electrolyte Balance and the EKG

As nurses we certainly know the importance of maintaining fluid and electrolyte balance in all patients under our care. Those patients with a cardiovascular disease are even more vulnerable to imbalances of fluids and electrolytes. It is not surprising that some of the treatments prescribed for patients can, in fact, cause electrolyte disturbances. Diuretics, dialysis, and such treatments can cause an insidious shift of electrolytes.

The two most important electrolytes are potassium and calcium. They are both vitally concerned with proper function of the cardiac muscle. Both electrolytes are needed in the proper amounts for contraction of the muscle and for the proper conduction of ht impulses through the special conduction pathway in the heart.

The nurse should be aware of the possibility of electrolyte imbalance. The nurse can then prevent serous clinical complications from occurring. Prevention is a prime nursing responsibility when caring for a patient with cardiovascular disease. In the text of EKG interpretation, we have to maintain the following nursing responsibilities concerned with maintenance of the normal EKG.

Hypokalemia (low potassium) is probably seen more than any other electrolyte disturbance. It can occur with vomiting, diarrhea, prolonged digitalis therapy, and prolonged nasogastic suctioning. These situations are common in acute hospitals. The nurse MUST be away that

electrolyte disturbances are insidious, and you must always keep a high awareness level. It may be difficult to do this when most nurses are concerned with the immediate problems of the patient. But always remember that there are long-term effects of n/g suctioning and prolonged vomiting and many other similar situations.

If a U wave is present on an EKG, the nurse should be alert to the fact that hypokalemia **may** be present. The U wave is normal in some people, but it can be indicative of an electrolyte disturbance. It is best seen in lead V(INSERT LITTLE 3) and is usually quite distinguishable unless it is hidden on the T wave.

If the U wave is hidden, it may give the T wave a notched appearance or make it prolonged. As the potassium depletion increases, the U wave becomes larger and more visible and the T wave becomes less visible. It may even become completely flattened or inverted.

If the hypokalemia continues, PVC's, PAC's, Junctional tachycardia, or even Ventricular tachycardia can occur. Any of these arrhythmias can lead to Ventricular fibrillation and death if left untreated.

Nursing Intervention = Early Recognition of the Problem

Hyperkalemia usually causes a change in the T wave. It is described as "peaked" or "tall." Hyperkalemia is not as common as hypokalemia. Normal T wave height is 5mm to 10 mm. In severe hyperalemia, A-V nodal block can occur and then untreated may lead to V Fib and death.

Calcium is the other important electrolyte mentioned earlier. It increased contractility of ht e4myocardium and, in fact, is essential to the conductivity of impulse on the myocardium. Hypocalcaemia and hyperalemia both can usually be detected on EKG by examining the Q-T interval.

Hypocalcaemia	**Hyperkalemia**
Renal failure, hyperparathyroidism, malabsorption syndromes, lengthened QT interval, lowered or inverted T wave	Hyperparathyroidism, neoplastic disease, acute osteoporosis, shortened QT interval, U waves may be present.

The QT interval is measured from the beginning of the QRS to the end of the T wave. The normal length of the QT interval will vary from person to person, but a general guide is: AT interval – ½ the R-R interval (when rate is 69-90 bpm).

In summary: fluid and electrolyte balance is important for maintenance of proper cardiac function. The nurse should be alert to changes in the EKG which can indicate f/e imbalance; OR if an imbalance is present already, the nurse should investigate the possible ramifications to the EKG.

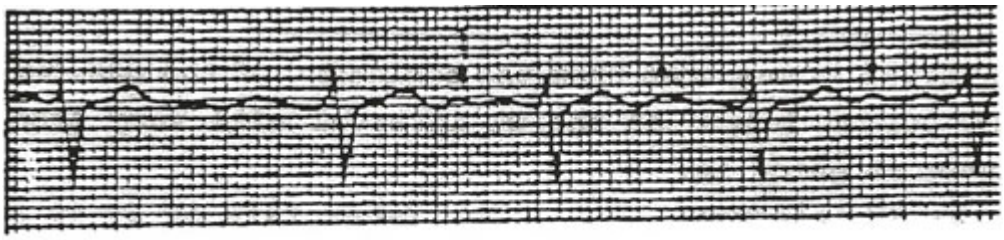
If you are a nurse in a critical care area, you already know the rapidity with which these changes can occur. You also have been trained to respond quickly to the emergency situation.

If you are a med/surg nurse, or other specialty nurse, be aware of imbalance which can happen slowly. Due to the insidious nature of most fluid and electrolyte problems, you must prevent it from becoming severe. Follow the steps below for prevention:

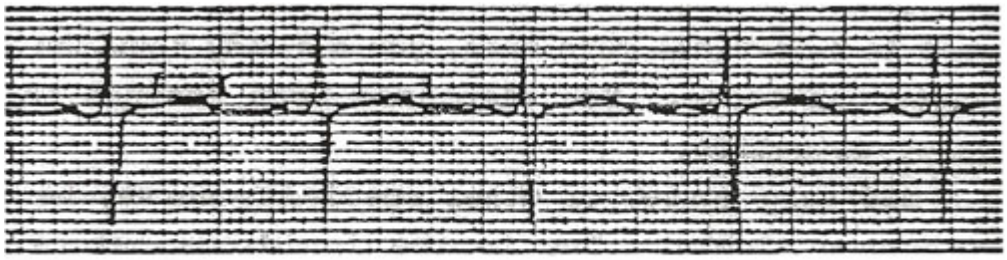
A. Recognition of Danger Situations
Patients on diuretics, cardiotonics, n/g suctioning, prolonged vomiting, etc.

B. Evaluate Fluid and Electrolyte Status
Investigate lab data, assess patient's symptoms, obtain EKG and rhythm strip.

C. Intervention
1. Inform MD of possible f/e disturbance.
2. Diet changes may be necessary (consult MD).
3. Develop nursing care plan to prevent further depletions of vital electrolytes; for example, comfort measures nutrition, medications, etc.

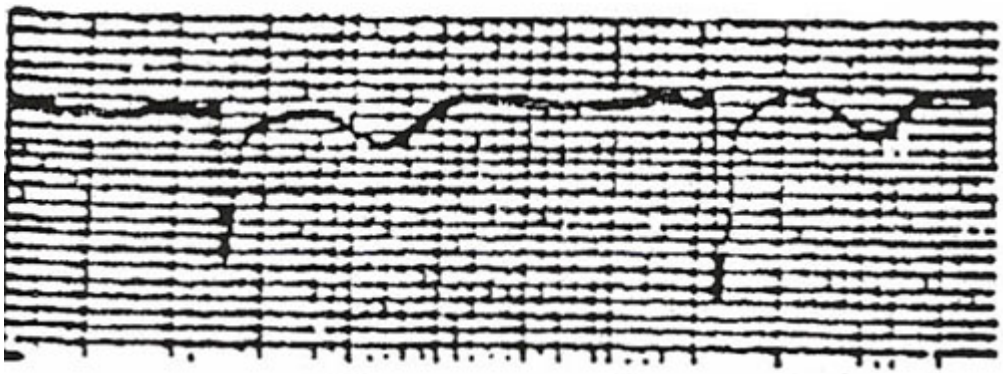


U waves are indicated here

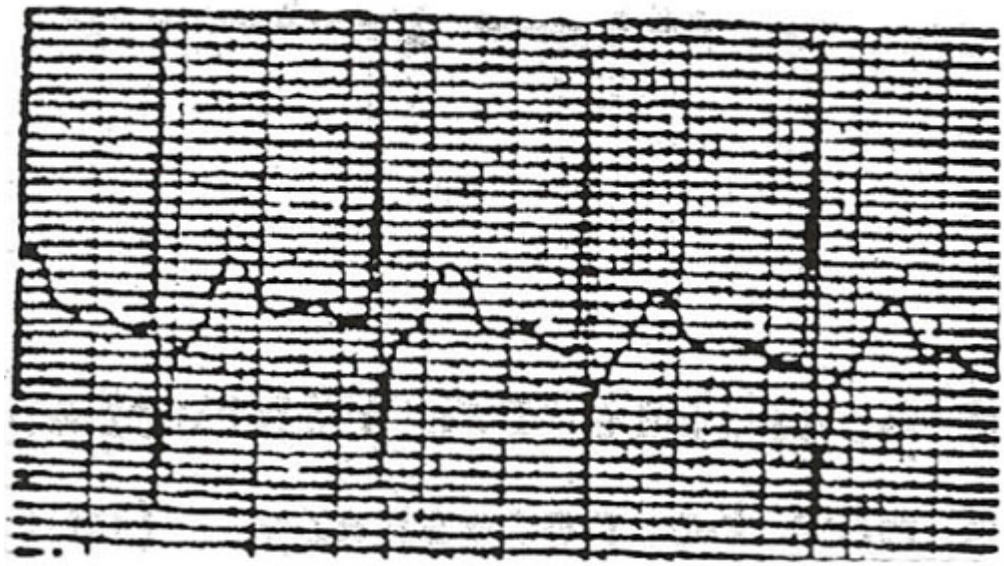


T waves and U waves are fused

Hypocalcemia



Hypercalcemia



Introduction to Heart Murmurs

When studying the basics of EKG, it is important that the nurse have an understanding of some of the aspects of the physical assessment of the cardiovascular system. It is not enough to study the interpretation of the EKG without paying attention to the related physical exam.

There is a definite relationship between the EKG and the patient's physical status. Some abnormal EKG patterns can cause no symptoms for one patient. However, the same arrhythmia

may cause devastating effects on another patient. Each person is different and the nurse must thoroughly assess what, if any, physical signs/symptoms the patient may exhibit.

Heart murmurs can be a significant finding by the nurse. Certainly the MD will assess the patient, however, some murmurs are intermittent and the MD may not discover them. The assessment by the nurse is important for an on-going status review of the patient and for discovering any possible changes in their conditions.

Heart Murmurs

Most commonly, heart murmurs are associated with abnormalities of the heart valves. The term murmur is a general term describing an abnormal sound in the cardiac cycle. It is an audible vibration caused by such things as a faulty valve and/or abnormal flow of blood. For example, blood flowing through the heart at a high rate alone may cause a murmur. Blood flow through a constricted valve and blood flow backward through incompetent valves can also cause a murmur. When ausculating heart sounds, the nurse should be aware of any abnormal sounds in the cardiac cycle. Also pay attention to the findings of the physical assessment which pertain to the cardiovascular system.

When assessing heart murmurs, listen for the following characteristics:

- Intensity.
- Timing: in the cardiac cycle.
- Loudness.
- Location.
- Radiation.
- Pitch.
- Quality.

Specific physical findings which are important:

- Under which conditions the sound is best heard.
- Changes during respirations.
- Postural changes which influenced the sound.
- Other assessments, skin, extremities, etc.

When ausculating a murmur, the nurse should try to relate it to the cycle of S^1 and S_2, the first and second heart sounds. S^1 is the sound of the onset of ventricular contractions when the mitral and tricuspid valves close. S_2 is the sound at the conclusion of the ventricular contraction when the aortic and pulmonic valves close. The interval between S^1 and S_2 corresponds to the systolic phase. The interval from S_2 to the S^1 corresponds to the diastolic phase of the cardiac cycle.

A murmur, then, that is heard between S^1 and S_2 is called a systolic murmur. A diastolic murmur is described as being between the S_2 to the beginning of S^1.

There is a way to more exactly define a murmur. The timing of the murmur can be defined as to the onset and duration. S systolic murmur may be described as early systolic, midsystolic, or late systolic. If the murmur is heard throughout the entire systolic phase it is called holosystolic or pansystolic.

Diastolic murmurs may also be described in the same manner as: early diastolic, middiastolic, late diastolic, or holodiastilic. A murmur that begins in the late diastolic phase and continues until the start of the systolic in commonly described as a presystolic murmur.

The next phase of assessing a murmur is how hit sounds. The characteristics you will listen for are:

- Loudness.
- Intensity.
- Quality.
- Pitch.

The loudness is graded on a scale of numbers. Variations from this scale are common in different regions of the country. However, it is agreed that a Grade I is the softest sound and a Grade V or Grade VI is the loudest sound. Some authorities use only Grade I through Grade III. Always become familiar with the system used at your facility.

There also exist patterns of intensity for heart murmurs:

1. Crescendo	Sound progressively increased in loudness.
2. Decrescendo	Sound progressively decreased in loudness.
3. Crescendo	Sound progressively increase, reaches maximum, decrescendo progressively decreases.
4. Plateau-Like	Sound maintains constant loudness.

The quality of the heart murmur may also be described as:

- Harsh.
- Musical.
- Blowing.
- Rumbling.

In summary: heart murmurs can be carefully described according to timing, loudness, pitch, and other characteristics. The nurse will have the responsibility for interpreting the EKG, and will also need to carefully assess all aspects of the patient's cardiovascular system including murmurs.

Use the following as a guide:

a. Timing _____
b. Intensity _____
c. Location _____

d. Radiation _____
e. Pitch _____
f. Quality _____
g. Location _____

Congestive Heart Failure (Cardiac Decompensation)

Definition:

Congestive Heart Failure (CHF) is described as cardiac decompensation. The heart decompensates after years of "compensating" for other medical conditions in the body that put a strain on the heart. DHF may also be caused by and acute disease, happing in a relatively short span. However, most cases of CHF, decompensation, develop over a period of years.

- Heart fails to meet oxygen demands of the body.
- Reduced cardiac output (relative to demands of the body cells).
- "Forward Failure" can't pump enough blood out.
- "Backward Failure" blood backs up due to inefficient pump.

Common Causes:

- Hypertension (over many years).
- Coronary Heart Disease.
- Valvular disease (usually aortic).
- Arteriosclerotic Heart Disease (ASHD).
- Congenital heart lesions.

Three mechanisms producing failure states:

- Failure to Fill.
- Overloading.
- Deterioration in functional capacity.

Nursing Assessment:

- Ausucultate heart sounds (Murmurs, S3, S4).
- Auscultate lung sounds (rales rhonchi).
- Assess arterial pressure (less than 90mmHg, continue frequent checks).
- Assess renal function – lowest accepted filtration is 20 – 30 cc per hour).
- Assess for distended neck veins, ascites, dependent edema.
- Assess liver engorgement and tenderness.
- Assess CNA – check sensorium, orientation, etc.
- Assess general circulation (eyes, ear lobes, tip of tongue, capillary refill, warmth of extremities, etc.)

Possible cardiac complications and arrhythmias:

- Gallop rhythm.
- Pulmonary edema.
- Sinus tachycardia.
- Ventricular tachycardia.
- PVC's

Chapter V The 12-Lead EKG

This chapter presents an introduction to the 12-lead ECG. The 12-lead ECG gives a tracing from 12 different "electrical positions" of the heart. Each lead is meant to pick up electrical activity from a different position on the heart muscle. This allows an experienced interpreter to see the heart from many different angles. This section is meant only as an introduction to the 12-lead ECG. It will take much practice of you to be able to interpret a 12-lead ECG tracing. This section will give you a basic understanding of how to take a 12-lead EKG, how to place the leads, and how to begin to interpret the tracing.

The electrocardiogram is a graphic record of the direction and magnitude of the electrical activity generated by the depolarization and repolarization of the atria and ventricles of the heart. This electrical activity is readily detected by electrodes attached to the skin. However, neither the electrical activity that results from the generation and transmission of electrical impulse, nor the mechanical contractions or relaxations of the atria and ventricles appear in the electrocardiogram.

An EKG lead consists of two surface electrodes of opposite polarity (one positive and one negative) or one positive surface electrode and a reference point. A lead composed of two electrodes of opposite polarity is called bipolar lead. A lead composed of a single positive electrode and a reference point is a unipolar lead.

For a routine analysis of the heart's electrical activity an ECG recorded from 12 separate leads is used. A 12-lead ECG consists of three bipolar limb leads (I, II, and III), the unipolar limb leads (AVR, AVL, and AVF), and six unipolar chest leads, also called precordial or V leads, (V_1, V_2, V_3, V_4, V_5, V_6).

Limb leads: I, II, III, IV, V, and VI
Lead IV also called AVR
Lead V also called AVL
Lead VI also called AVF

Chest leads: V_1, V_2, V_3, V_4, V_5, V_6

Below is a sample of a 12-lead EKG tracing.

It is a normal tracing (shows normal sinus rhythm). This course is meant to give the nurse a practical education concerning the 12-lead EKG. We will present the basic clinical aspects of the 12-lead. We will demonstrate how and where to attach the leads in certain places. We will also demonstrate the basics of interpreting the results. Then all you need is practice.

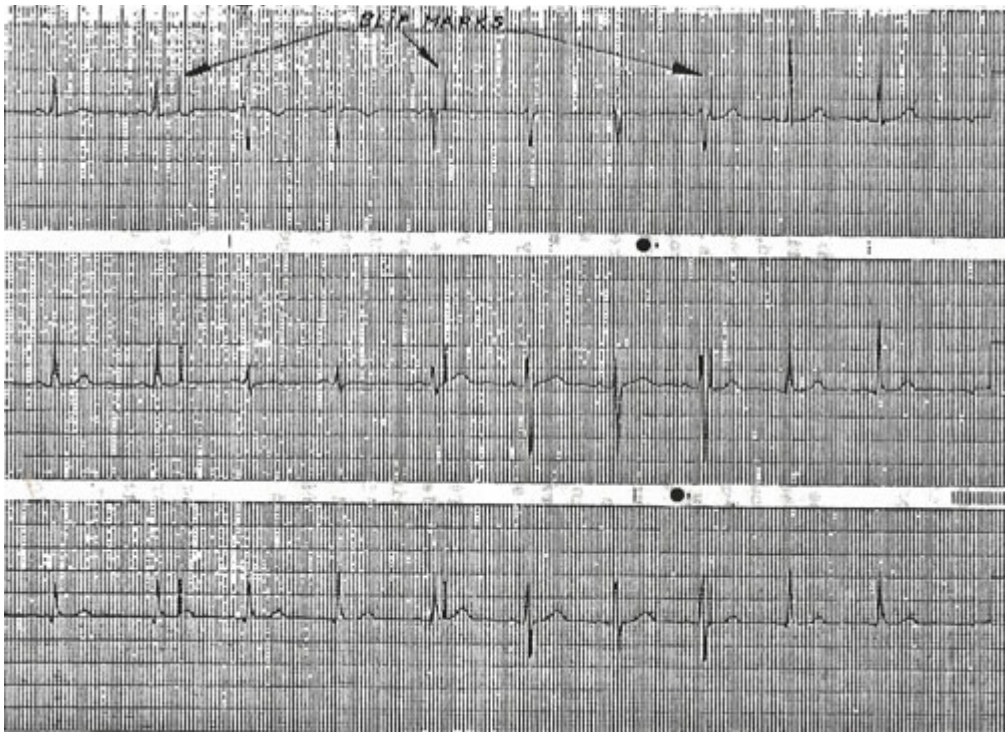
Each 12-lead EKG machine will have its own instructions for use. Be sure you are familiar with the machines at your facility. The instructions for the machine will show you how to attach the leads to the patient. In most instances, the patient electrodes will be attached with the use of either flat elastic straps or by cloth Velcro straps. In most cases, the metal electrodes will also need to be coated with conductive gel prior to attachment to the patient. Be sure to clean the electrodes before and after each use, as gel will tend to build up. Be sure to read the entire instruction manual for the machine including: how to start the machine, how to load the paper, how to calibrate the machine (if needed), and any other pertinent information needed to safely and accurately run the machine.

Once you know how to operate the machine at your facility the next concept to understand is the placement of the leads. The limb leads are usually first. As the name implies, the limb leads are attached to the four limbs. This is usually accomplished by attaching the leads, according to instructions, on each wrist and each ankle. As mentioned earlier, the electrodes will be sensing the electrical impulses from the heart muscle at ht various locations and with various voltages, either positive or negative.

The 12-lead EKG tracing below was obtained with universal lead placement. “Blip Marks” are pointed out on the tracing. These marks are for the purpose of showing the leads as they are changed. Every time you see a blip mark, the next lead is being recorded on the tracing.

The placement is as follows:

Lead I through lead 6	Limb leads
Lead 7 through lead 12	Chest Leads



Location of the Frontal Plane Axis:
In order to accurately interpret the 12-lead EKG, you must have an understanding of the electrical activity of the heart. The direction in which the impulses flow in the heart is important. It is also important to understand that 12 different leads pick up those impulses as they travel in many different directions through the heart.

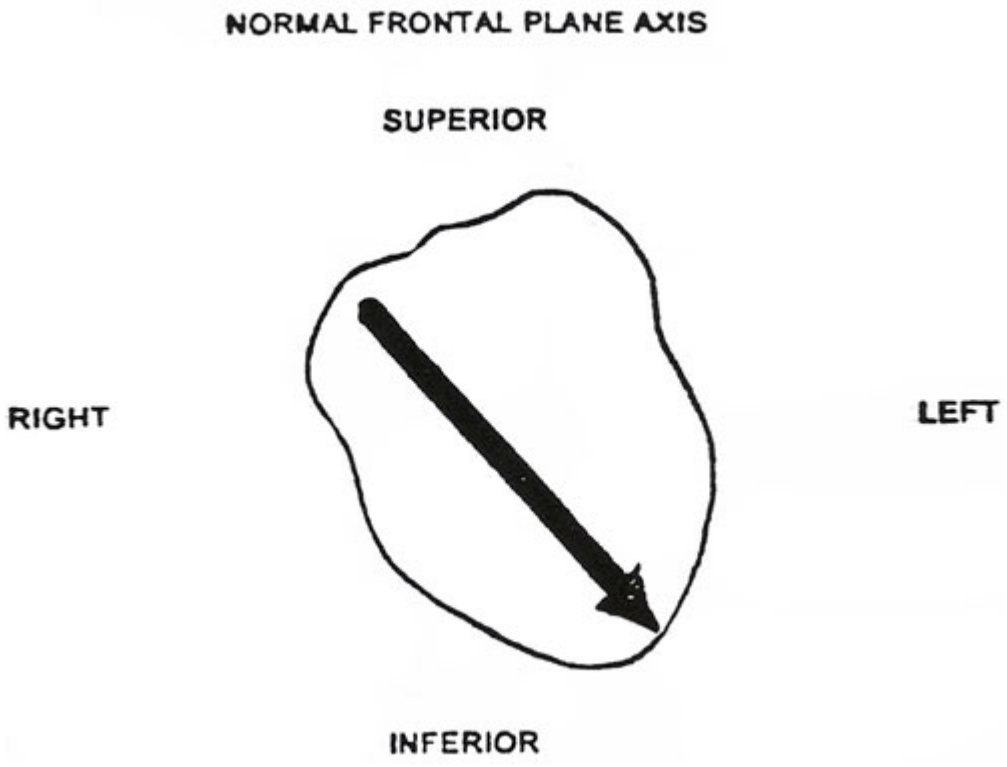
Definition:

The frontal plane axis is the orientation of the heart's electrical activity in the frontal plane.

The fontal plane consists of:

Right-to-Left	/	Left-to-Right Directions	AND:
Superior-to-Inferior	/	Inferior-to-Superior Directions	

*See illustration below.

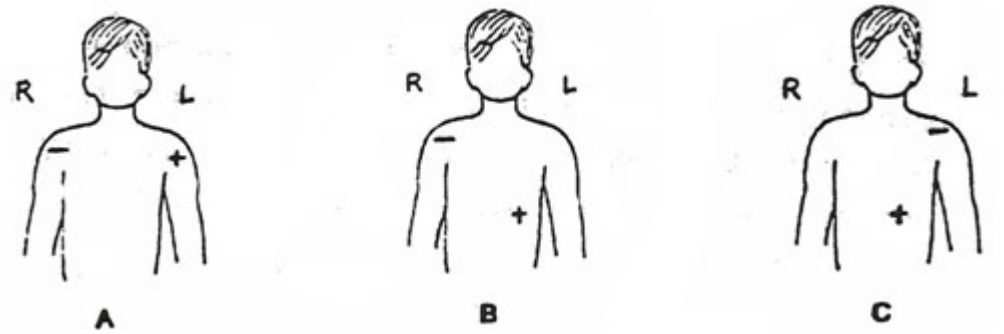


Remember that the normal conduction of the heart begins in the SA node. The wave of depolarization moves across the atria, through the AV node, into the Bundle of HIS, down the Bundle Branches, and finally through the Purkinje fibers which conduct the electrical impulses throughout the ventricles.

The Frontal Plane Leads:

The activity of the heart produces electrical potentials that can be measured on the surface of the skin. Using the galvanometer (EKG machine), differences between electrical potentials at different sites of the body can be recorded.

See illustration below:



In picture A above, the negative electrode is on the right arm and the positive electrode is on the left arm. This is lead I. Lead I records electrical difference between the left and right arm electrodes.

In picture B above, the negative electrode is on the right arm and the positive electrode is on the left leg (left lower chest). This is lead II. Lead II records electrical differences between the left leg and right arm electrodes.

In picture C, the negative electrode is on the left arm and the positive electrode is on the left leg (left lower chest). Picture C depicts lead III. Lead III records electrical difference between the left leg and the left arm electrodes.

The above illustration shows Leads I, II, and III, their placement and the electrical potential on these three leads.

They are summarized as follows:

Lead I: Right arm-negative, Left arm-positive

Records electrical differences between the left and right arm electrodes.

Lead II: Right arm-negative, Left leg-positive

Records electrical difference between the left leg and right arm electrodes.

Lead III: Left arm-negative, Left leg-positive

Records electrical differences between the left leg and left arm electrodes.

The other three frontal plane limb leads are called the augmented Vector leads. The Galvanometer (EKG machine) records potential differences and, therefore, the technique is Bipolar (potential site A minus potential site B). However, if the potential of B is zero the recorder records only the potential site A. this means that these next three electrodes, for all practical purposes have a zero potential and do not change during the cardiac cycle. They became known as the V electrodes, and all three leads became known as the V electrodes or UNIPOLAR leads.

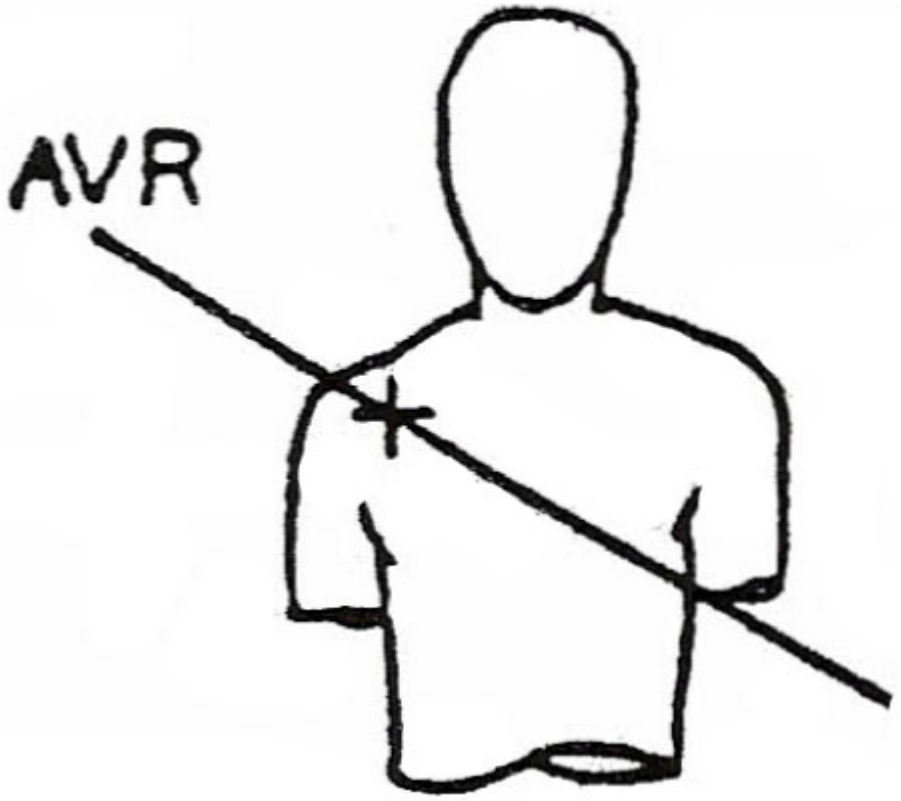
As mentioned earlier, unipolar leads measure the electric impulses at only one point, instead of across two points, as the first three leads. With these V leads, the second site is -0- so there is no need to measure from two pointes, only one point is needed. To obtain the measurements from these V leads, you simply turn the dial on the EKG machine to aVR, aVL, and aVF, respectively. The machine automatically makes the needed connection to measure the voltage from these areas.

The illustrations below show where these measurements take place.

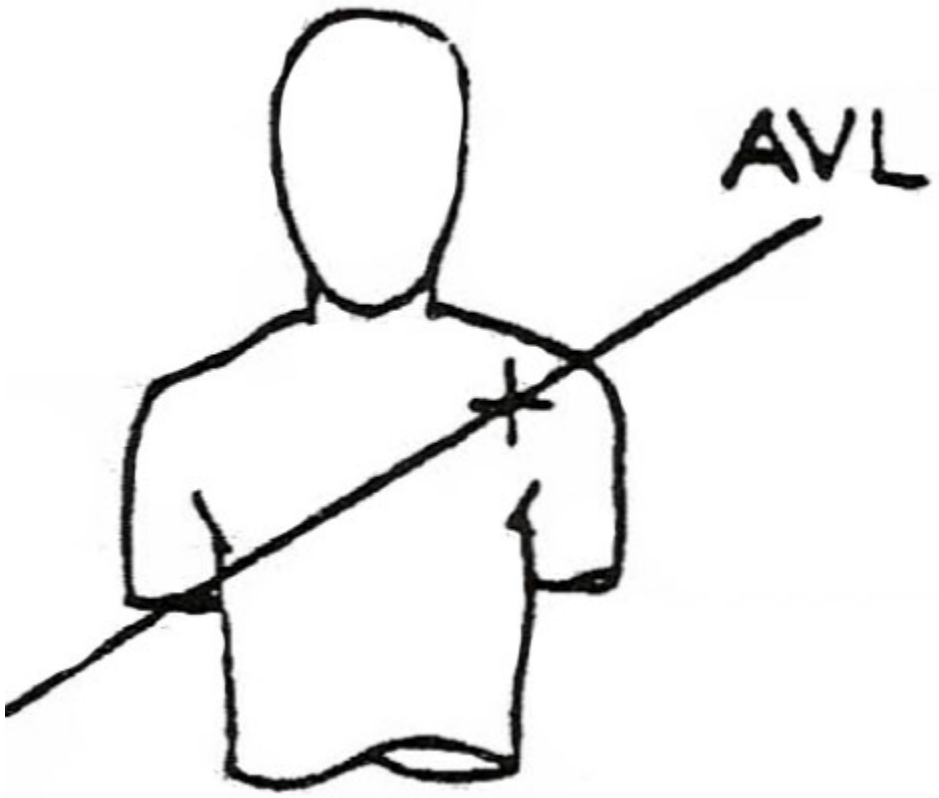
They are summarized here:

Lead aVR Augmented Vector Right, positive electrode right shoulder.
Lead aVL Augmented Vector Left, positive electrode left shoulder.
Lead aVF Augmented Vector Foot, positive electrode on Food.

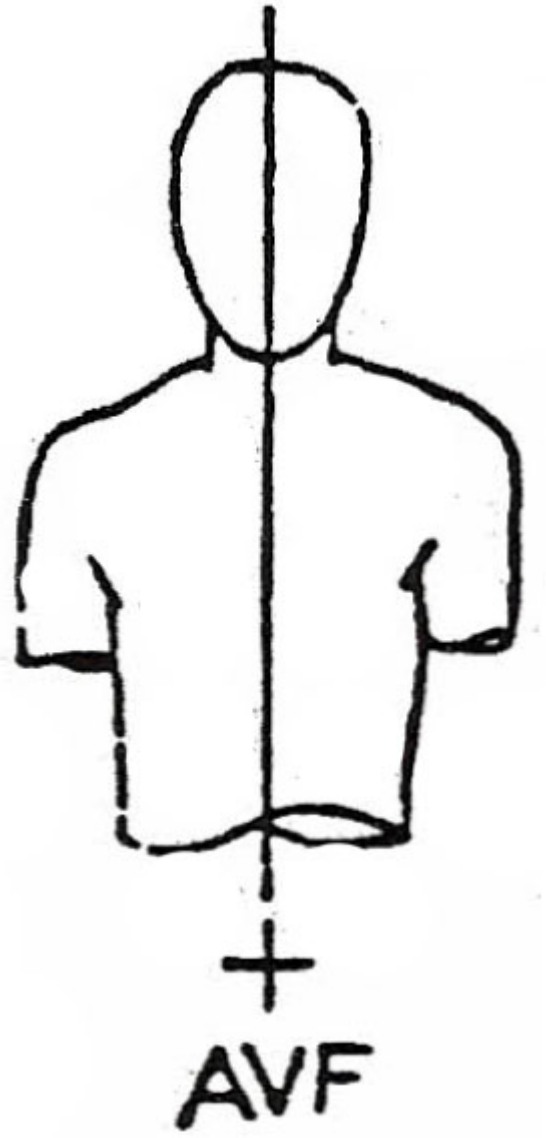
aVR means augmented Vector Right; the positive electrode is on the right shoulder.



aVL means augmented Vector Left; the positive electrode is on the left shoulder.

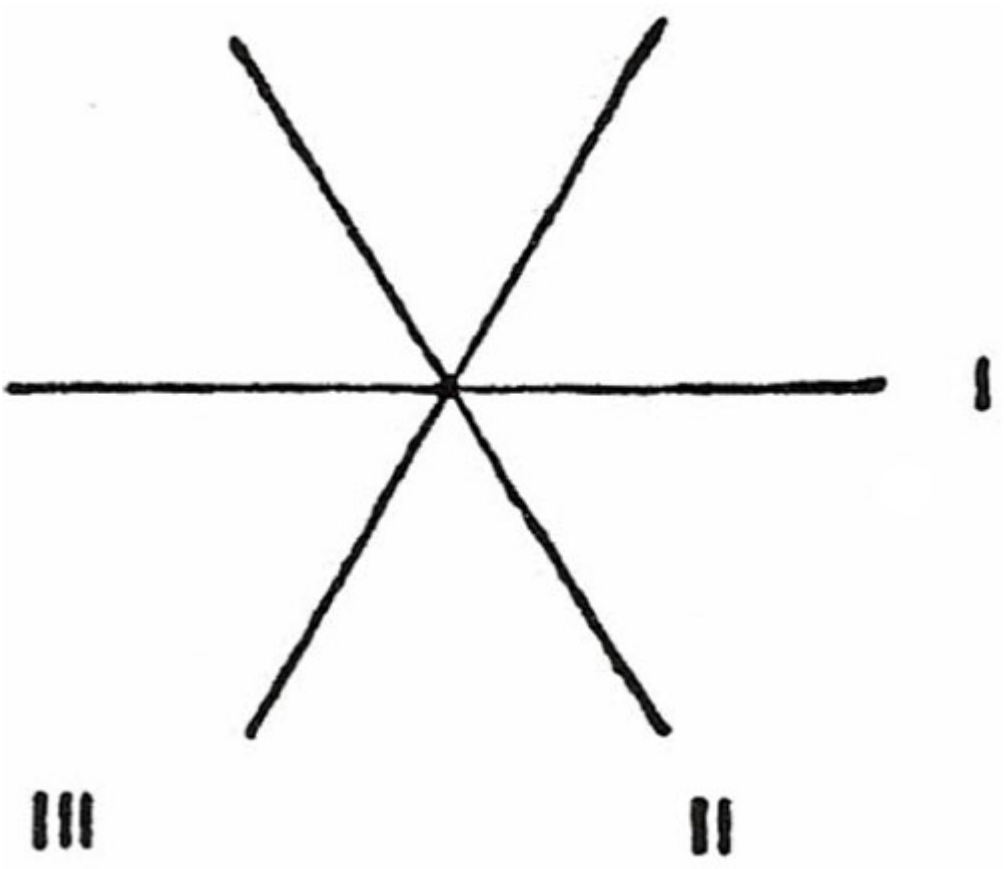


aVF means augmented Vector Foot; the positive electrode is on the foot.

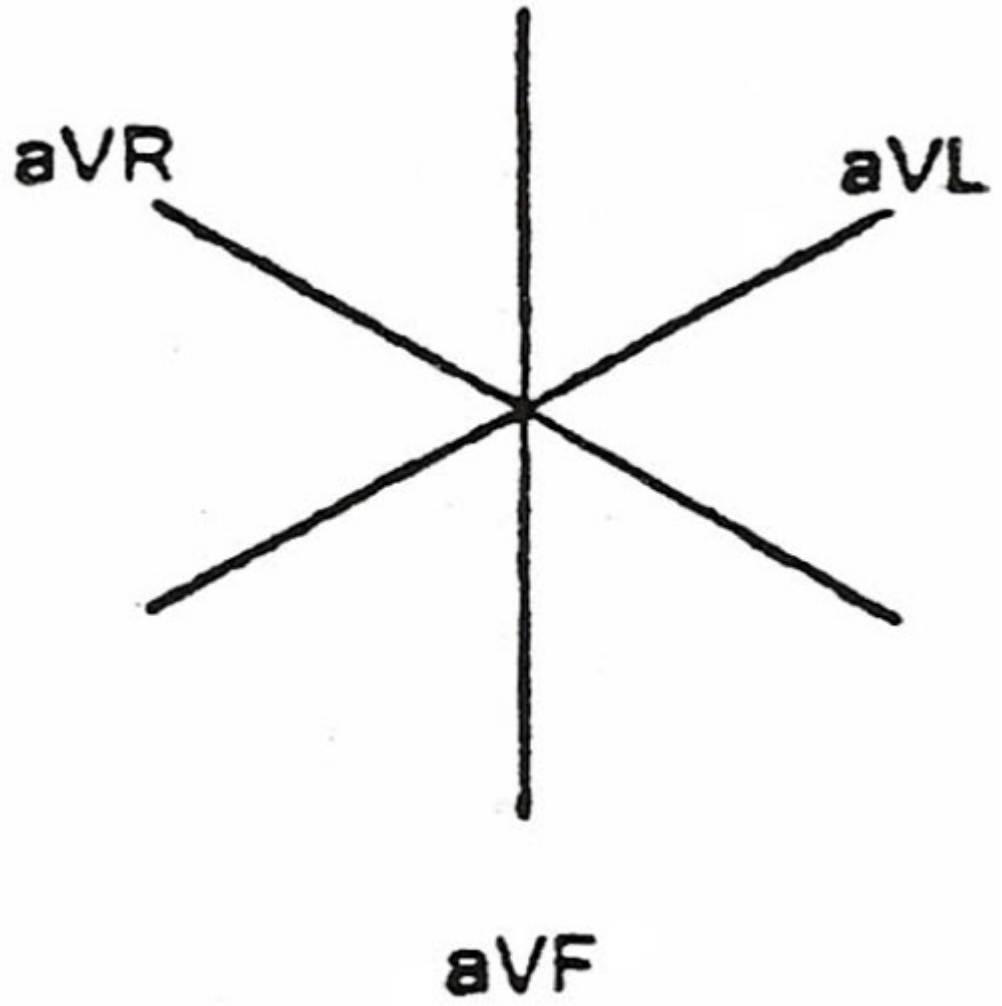


NOTE: although the F stands for foot, please conceptualize the positive electrode of aVF as being at the umbilicus.

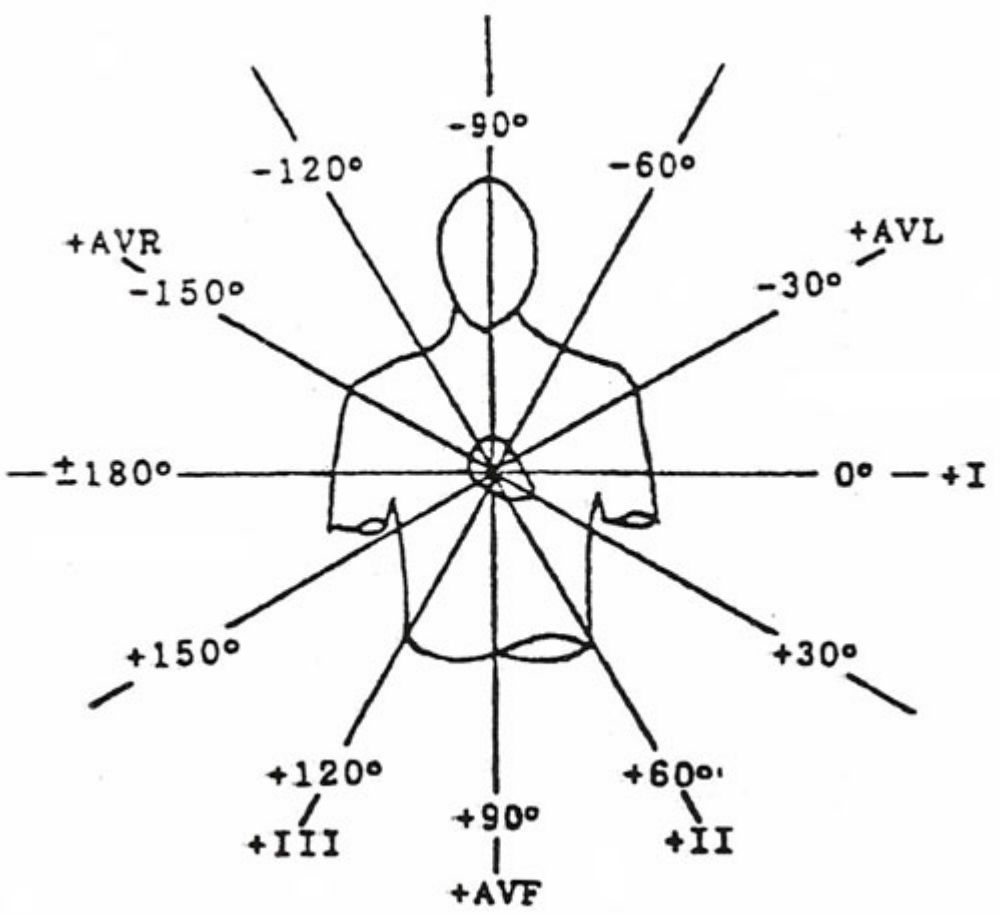
Now combine the three limb leads I, II, III



And the three augmented Vector leads aVR, aVL, aVF



and this combination creates the Hexaxial Reference System



As the above illustrations point out, the six limb leads measure the electrical activity of the heart from the frontal plan. The frontal plane only manes that the patient is in anatomical position and facing you. Therefore, the patient's left side is on your right side.

The six limb leads measure a copulate circle or 360 degrees around the heart. They measure the electrical activity of the heart from every possible angle. The reason for this is obvious. By measuring he heart from different angles, you will be able to pinpoint the location of any conduction deft in the heart.

If you refer to the previous illustration you will notice that 306 degrees of the front of the heart are completely covered by the six limb leads. This six fontal plane leads placed across the heart form the hex axial reference system. This system is the means by which we communicate the location of the frontal plane axis.

The angles are as follows;

LEAD I	Is located at 0 degrees	And	(+) (-) 180 degrees
LEAD II	Is located at +60 degrees	And	-120 degrees
LEAD III	Is located at +120 degrees	And	-60 degrees
LEAD aVR	Is located at +30 degrees	And	-150 degrees
LEAD aVL	Is located at -30 degrees	And	+150 degrees
LEAD aVF	Is located at +90 degrees	And	-90 degrees

*NOTE: the (+) positive and (-) negative designation on these degrees of each lead DO NOT RELATE TO THE (+) AND (-) ELECTRODES. Note also that all the positive (+) degrees are on the inferior surface of the hexaxial figure. All the negative (-) degrees are on the superior surface of the hexaxial figure. In the future, you may wish to study in more detail the interpretation of the tracings made in each of these six leads. In this course we have gone into detail of the lead II interpretation only.

The Chest Leads (or Precordial Leads)

We will now discuss the remaining six leads of the 12-lead EKG. These next six leads are called the Chest leads or the Precordial leads. This part may also become confusing because these precordial leads are six additional leads that use the same V technique we used with the 3 limb leads. Therefore, do not confuse these Precordial V leads with the three V limb leads (aVR, aVL, aVF).

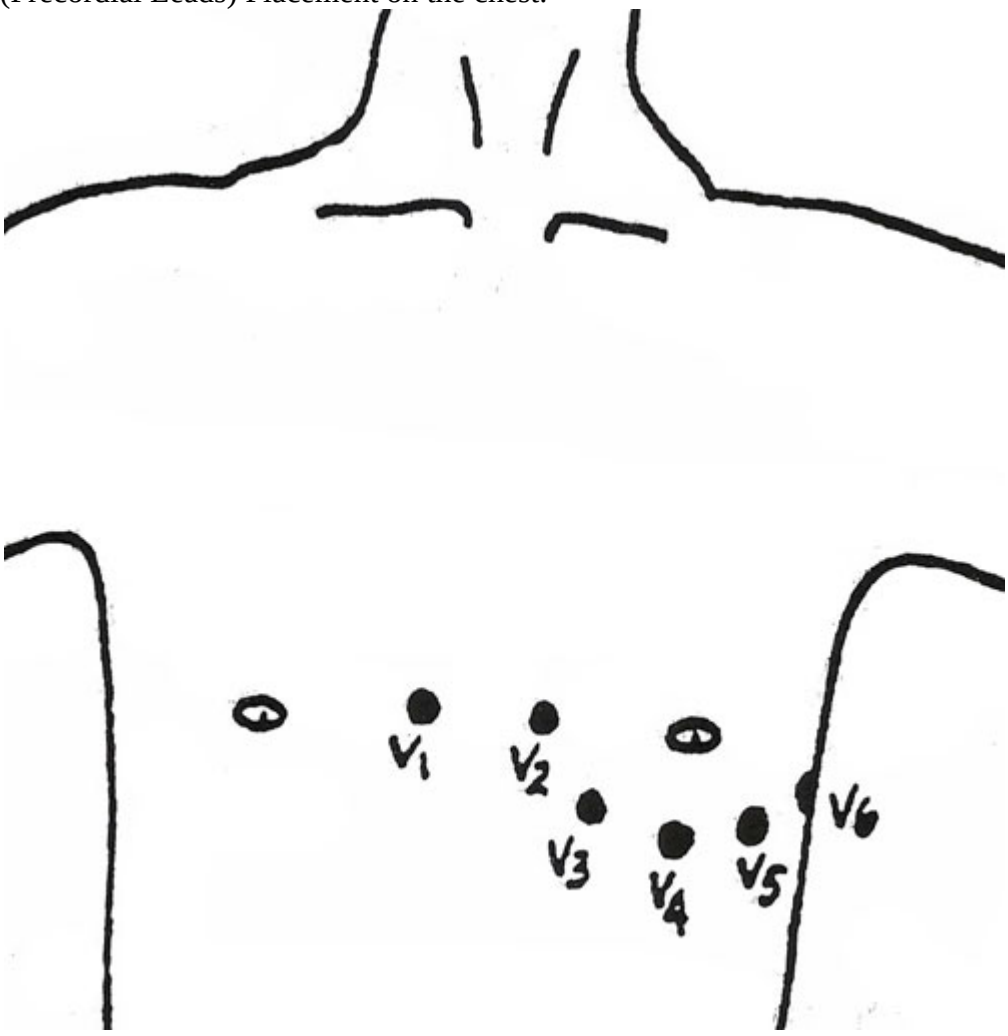
The precordial (chest leads) leads each consist of a positive electrode strategically placed on the chest of the patient. The positions of the positive electrode for the six precordial leads are very important for a valid tracing to be made on the EKG machine.

These positions are: V_1, V_2, V_3, V_4, V_5, V_6

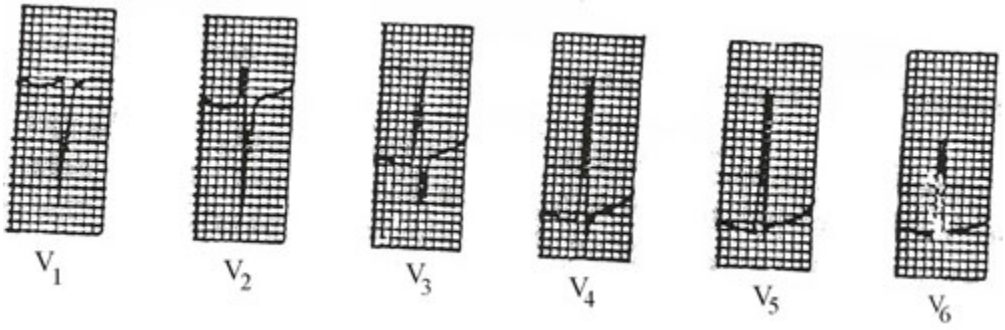
V_1	positioned:	Fourth intercostals space, right sterna border.
V_2	positioned:	Fourth intercostals space, left sterna border.
V_3	positioned:	One-half way between V and V in straight line with them.
V_4	positioned:	Fifth intercostals space, left midclavicular line.
V_5	positioned:	Fifth intercostals space, left anterior auxiliary line.
V_6	positioned:	Fifth intercostals space, left midauxiliary line.

These positions are critical for interpretation of the EKG. At fort it may seem complicated to position these electrodes. After some practice it is relatively easy to place the leads properly. The below illustration will show the correct positions across the chest. One of the main reasons that the precordial leads are important is that these leads show the R Wave Progression. From V (NUMBER) through V (NUMBER) the R Wave becomes progressively larger. The experienced interpreter of these leads will be able to rule out many different cardiac disorders by attaching the R wave and other configurations in the pericardial leads.

V - Lead (Precordial Leads) Placement on the chest.



The normal ECG morphology of the complex in the V leads:



Conclusion

The unipolar of V lead technique we just presented is used on a horizontal plane with the chest or precordial leads. The precordial leads consist of a positive electrode strategically placed on the chest. The positions of these precordial leads are shown in the preceding illustration. These chest leads detect electrical impulses from the heart on a horizontal plane across the heart. This tracing gives a different view of the heart and allows detection of arrhythmias that are undetectable from the frontal plane angles.

The first six leads of the 12-lead EKG are obtained using the frontal plane technique. This frontal plan detects electrical impulses as they follow the nurse pathways along the frontal plane of the heart. Remember that the objective of taking a 12-lead EKG is to be able to "look at the heart" from different angles. You get a different "view" of the heart from the various leads that are used.

In the first chapters of this course we learned how to interpret the EKG and to detect he various arrhythmias. These earlier chapters were all focusing on Lead II. The 12-lead EKG gives more detailed record of the heart's electrical activity from many different angles. As we stared earlier, this chapter is just an introduction to the 12-lead EKG. You will need to take a more advanced course in EKG in order to interpret the results of an EKG tracing in all 12 leads.

We hope you have enjoyed this course and gained a practical knowledge of interpreting the EKG. We have presented a number of different samples of EKG tracings so you may practice on various types of tracings, not just one. When you first start to interpret EKG tracings, remember to follow the steps we listed in the previous chapter. Practice on strips you might find on your job. Go to your ICU and ask for sample strips to practice. The nurses and doctors there might even help you interpret them. Once you become more skilled, it will become easier to recognize arrhythmias without going through all the steps but as a beginner, you will want to carefully follow all the steps to be sure you don't overlook the dangerous arrhythmias. Remember to practice as much as possible and use other references for the more completed EKG tracings.

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EKG Interpretation Test

1. Outermost layer of the heart; consists of parietal and visceral layers?
    a. Epicardium.
    b. Pericardium.
    c. Myocardium.
    d. Epicardium.

2. The normal activity of the heart conducts impulses from the SA node to:
    a. Individual muscle cells.
    b. Groups of muscle cells.
    c. RA node.
    d. RP node.
    e. None of the above.

3. Muscle contraction is dependent upon the availability of calcium and:
    a. Other electrolytes.
    b. Other elements.
    c. Certain drugs.
    d. Digitalis only.

4. This is a symbolic representation of physical force?
    a. Vector.
    b. Plane.
    c. Membrane potential.
    d. Physical tension.

5. This is the state where the cell returns to its polarized state and the polarity reverses?
    a. Reverse polarity.
    b. Inverse polarity.
    c. Depolarization.
    d. Action potential.
    e. Repolarization.

6. _____ cells have one phase of action potential.
    a. Action cells.
    b. Pacemaker cells.
    c. Depolarized.
    d. Repolarized.

7. Normal flow of current in the heart is primarily from the base to the:
    a. Center.
    b. Apex.
    c. Middle.
    d. Care.
    e. Nucleus.

8. _____ lies in the wall of the right atrium near the inlet of the superior vena cava?
    a. AV node.
    b. Purkinje network.
    c. SA node.
    d. Bundle of HIS.

9. _____ records the time sequences and amplitude of the electrical activity of the heart.
    a. EKG machines.
    b. EKG monitors.
    c. EKG paper.
    d. EKG technician.

10. The onset of the QRS complex is indentified as the point where the first wave of the complex just begins to:
    a. Increase.
    b. Decrease.
    c. Deviate.
    d. Return to baseline.

11. _____ is the first positive deflection in the QRS complex.
    a. QRS complex.
    b. P Wave.
    c. S Wave.
    d. R Wave.
    e. U Wave.

12. The DURATION of the QRS complex is 0.06 to 0.10 second in adults.
    a. 1.00-1.20 second.
    b. 0.08 – 1.00 second.
    c. 0.12 – 0.20 second.
    d. 0.06 – 0.10 second.

13. _____ represents ventricular repolarization.
    a. QRS complex.
    b. T Wave.
    c. Q Wave.
    d. P Wave.
    e. U Wave.

14. A _____ always follows as QRS complex.
    a. QRS complex.
    b. T Wave.
    c. Q Wave.
    d. P Wave.
    e. U Wave.

15. A _____ indicates that repolarization of the ventricles has occurred.
    a. U Wave.
    b. T Wave.
    c. QRS complex.
    d. P Wave.
    e. T Wave.

16. A lead composed of two electrodes of opposite polarity is called a:
    a. Bipolar lead.
    b. Unipolar lead.
    c. Polarized lead.
    d. Repolarized lead.

17. The NSR implies that all of the beats have a normal:
    a. Pacemaker.
    b. Wave patter.
    c. Ectopic beats.
    d. R Wave.

18. When either the rate or the contour of any of the individual waves is abnormal, the disorder is called:
    a. Pacemaker rhythm.
    b. Ventricular rhythm.
    c. Escape beat.
    d. Inverted wave.
    e. Arrhythmia.

19. The rate increases during inspiration and then slows during expiration, this arrhythmia is called:
    a. Asystole.
    b. PVC's.
    c. PAC's.
    d. Sinus arrhythmia.
    e. Sinus tachycardia.

20. The simplest form of treatment for this arrhythmia may be the administration of a sedative or tranquilizer:
    a. PAT.
    b. Sinus tachycardia.
    c. Sinus arrhythmia.
    d. Ventricular fibrillation.

21. This arrhythmia is characterized by a prolonged conduction of the impulses through to the ventricles:
    a. Complete heart block.
    b. First degree block.
    c. Sinus arrest.
    d. Ventricular tachycardia.

22. In this type of second degree block, there is a progressive increase in the PR interval:
    a. Type A.
    b. Type B.
    c. Type I.
    d. Type II.
    e. Mobitz block.

23. This type of heart block is complete or total blockage of all impulses through the AV node:
    a. Type A.
    b. Type B.
    c. Type I.
    d. Second degree.
    e. Third degree.

24. This arrhythmia has delayed ventricular stimulation, usually due to blockage of impulses traveling through the bundle of HIS:
    a. First degree block.
    b. Second degree block.
    c. Third degree block.
    d. Mobitz block.
    e. Bundle branch block.

25. In this arrhythmia, the pacemaker of the heart shifts from the normal SA node to the atria or to the AV Junction.
    a. Wandering atrial pacemaker.
    b. Premature atrial contraction.
    c. Premature ventricular contraction.
    d. Ventricular pacemaker.

26. This arrhythmia has extra beats which occur from an ectopic focus on the ventricle wall.
    a. PAC's.
    b. PVC's.
    c. NBC's.
    d. PAT.
    e. None of these.

27. With this arrhythmia, the blood pressure drops immediately to zero and so does the cardiac output:
    a. PAC's.
    b. Atrial tachycardia.
    c. Ventricular fibrillation.
    d. Ventricular tachycardia.

28. This arrhythmia is the same as PVC's, except that there are many of them in a row.
    a. PAC's.
    b. PAT.
    c. Ventricular fibrillation.
    d. Atrial fibrillation.
    e. Ventricular tachycardia.

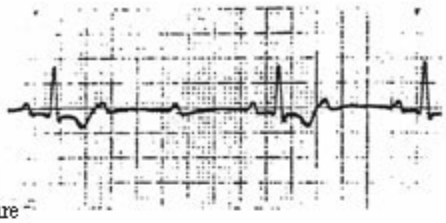
29. _____ Fibrillation is more likely to be reversed because it indicates a recent onset of fibrillation.
    a. Atrial.
    b. Ventricular.
    c. Fine.
    d. Early.
    e. Course.

30. This arrhythmia may occur in paroxysms of three or more PVC's separated by the underlying rhythm:
    a. Ventricular.
    b. Atrial fibrillation.
    c. Atrial flutter.

31. Escape rhythm refers to the automatic pacemaker of the heart located:
    a. SA node.
    b. Bundle branches.
    c. Atrial myocardium.
    d. Ventricles.

For questions 32 – 36 indentify the below arrhythmias.

Figure 1

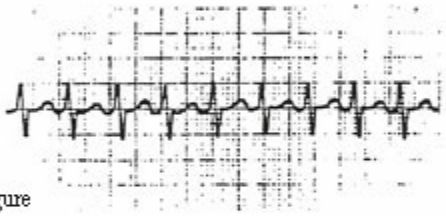


Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

Figure 2

Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

Figure 3

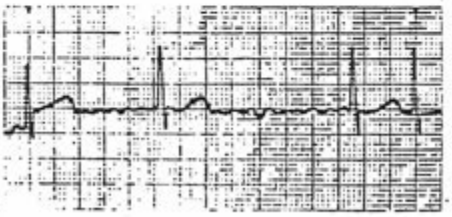


Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

Figure 4

Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

Figure 5



Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

32. Sinus Tachycardia:
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

33. Atrial fibrillation (fine):
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

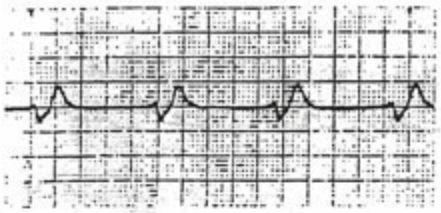
34. NSR with second-degree, 2:1 block:
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

35. Normal Sinus Rhythm
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

36. Supraventricular tachycardia:
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

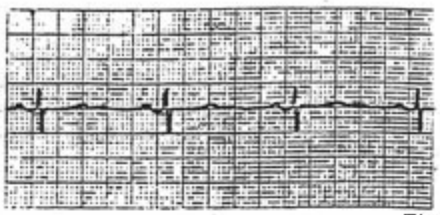
For questions 37 – 41 indentify the below arrhythmias.

Figure 1



Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

Figure 2

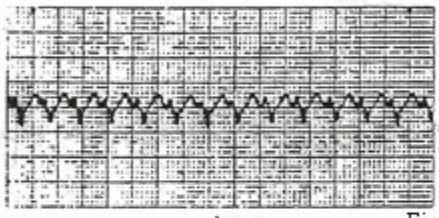


Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

Figure 3

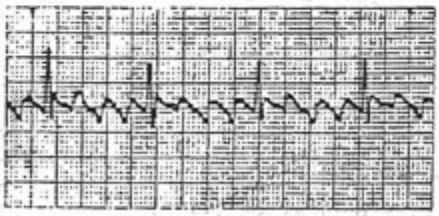
Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

Figure 4



Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

Figure 5



Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

37. Atrial tachycardia:
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

38. Accelerated junction rhythm (idoventricular):
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

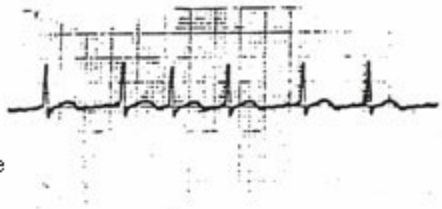
39. Ventricular fibrillation (fine):
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

40. Normal Sinus Rhythm:
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

41. Atrial flutter:
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

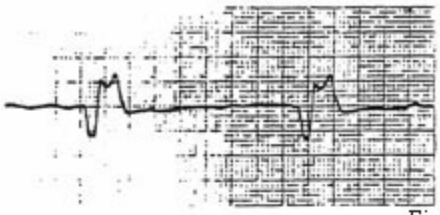
For questions 42 – 46 indentify the below arrhythmias:

Figure 1



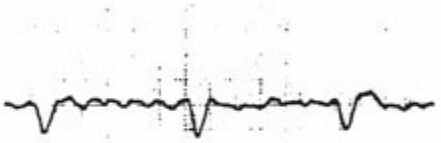
Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

Figure 2



Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

Figure 3

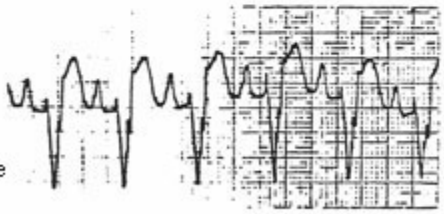


Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

Figure 4

Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

Figure 5



Rate: _____
Rhythm: _____
P Waves: _____
P-R Int: _____
QRS: _____
Intrp: _____

42. Ventricular tachycardia:
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

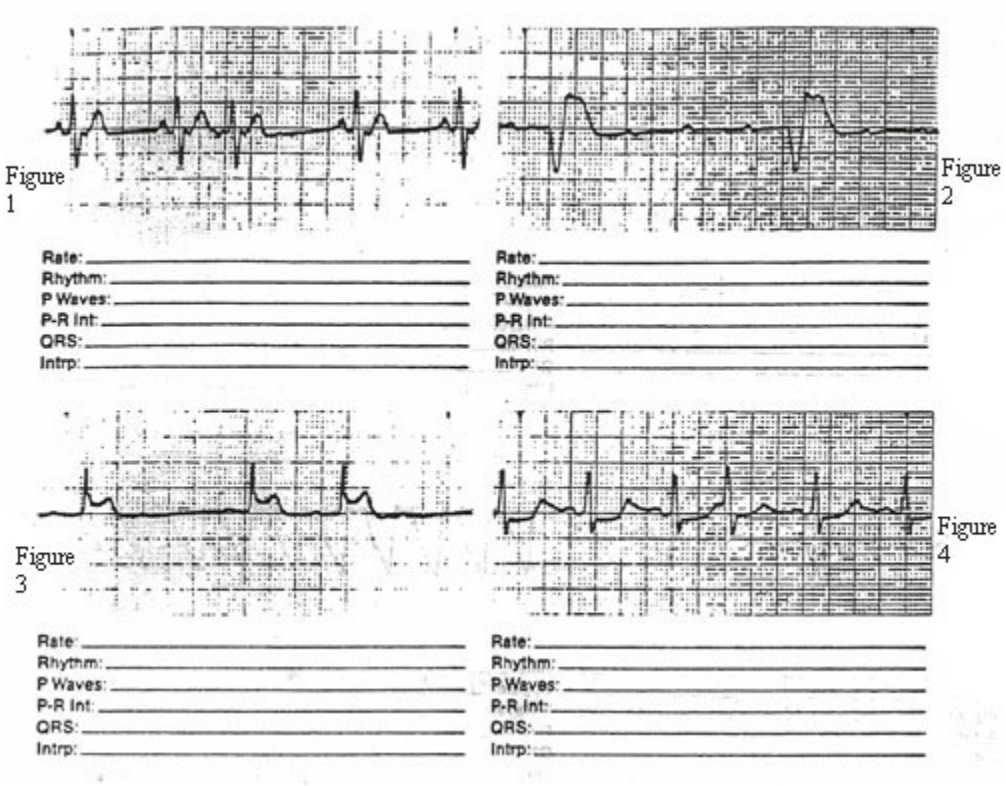
43. Ventricular Escape Rhythm:
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

44. Atrial flutter with BBB:
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

45. Atrial fibrillation (fine):
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

46. Sinus tachycardia with BBB:
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.
    e. Figure five.

For questions 47 – 49 indentify the below arrhythmias:



47. Third-degree AV Block with wide QRS complexes:
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.

48. NRS with Premature Junctional Contraction:
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.

49. Second Degree AV Block (Wenckebach):
    a. Figure one.
    b. Figure two.
    c. Figure three.
    d. Figure four.