ISSUED BY: ICU Patient Care Team
AUTHORIZED BY: Critical Care Program Council
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CATEGORY: Certification **PAGE:** 1 of 73

SUBJECT: BASIC ECG INTERPRETATION

DESIGNATION Advanced Nursing Skill

REQUIRED BY Registered Nurses in Critical Care Program (CVT MS ICU/SDU, Cardiac Medical Unit [CMU], Heart Failure Disease Management Program [HFDMP], Cardiodiagnostic, Cardiology, Cardiac Rehabilitation, Emergency Department [ED]

Certification Requirements

Prerequisites: **None**

1. **Hours of Instruction:**
   - Theoretical: 15.0 hours
   - Instructor: Critical Care Program Nurse Clinician and/or delegate

2. **Certifying Process:** Successful completion of written test and demonstration of knowledge during clinical testing for electrical defibrillation certification

3. **Certifying Personnel:** Critical Care Program Nurse Clinician or delegate

TECHNIQUE

1. The Registered Nurse seeking additional knowledge in ECG Interpretation in order to competently provide assessment/care for patients requiring cardiac monitoring shall attend the theoretical classes and successfully complete test.

2. The Registered Nurse seeking certification in electrical defibrillation, applying transcutaneous pacemaker, administering life-saving drugs, and adjusting temporary transvenous pacemaker shall
   - **a.** attend the theoretical classes
   - **b.** successfully complete test
   - **c.** successfully interpret ECG rhythm during practical tests in electrical defibrillation, applying transcutaneous pacemaker, life-saving drugs, and adjusting temporary transvenous pacemaker

BASIC ECG INTERPRETATION

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ASSOCIATED SCIENCE BASE

Anatomy and Physiology of the Heart

The heart is a hollow, muscular organ situated in the thorax with the lungs on either side and the diaphragm below it. The base of the heart lies parallel with the right edge of the sternum. The apex of the heart is at the mid-clavicular line at the 5th intercostal space. The heart is approximately the size of the individual's closed fist.

Layers of the Heart

The heart is contained within a fibrous sac called a pericardium. It is composed of a serous membrane and contains a fluid called pericardial fluid. This fluid prevents friction between the heart and the pericardium during pumping. There is usually 30-80 ml of fluid within the pericardial sac.

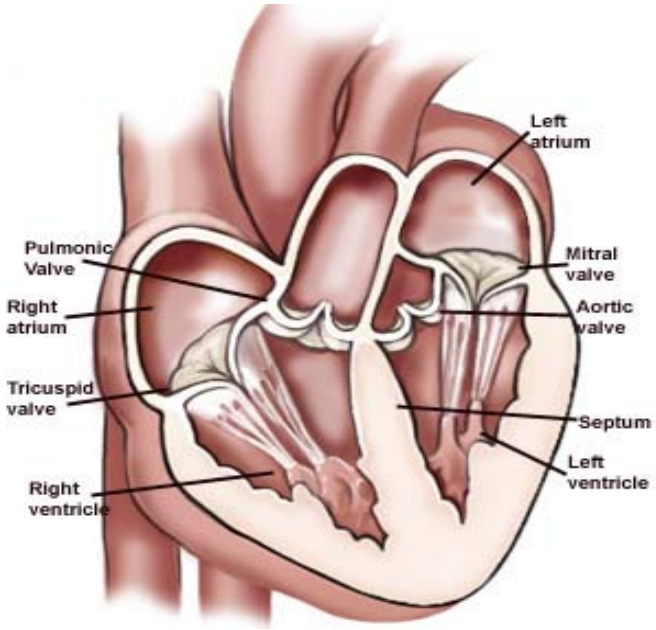
Epicardium -the outermost layer of the heart; composed of a serous membrane

Myocardium -the middle layer of the heart but the main component; composed of muscle

Endocardium -the inner surface of the heart; composed of endothelial cells and covers the valves,
-surrounds the chordae tendineae and is continuous with the lining membrane of the large vessels.

Cavities of the Heart

The heart is divided into a right and left side by the septum. Each side of the heart differs in function, musculature and valvular structure. Each side of the heart contains an upper cavity (atrium) and a lower cavity (ventricle). Both sides of the heart contract "almost simultaneously."

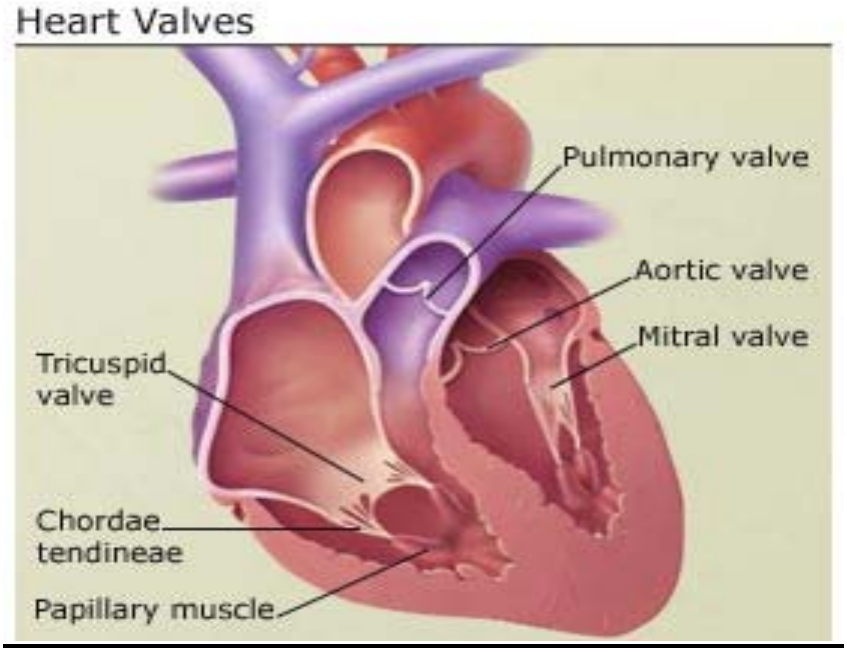


Valves of the Heart

The **tricuspid valve** is located between the right atrium and the right ventricle. It is composed of three cusps which are composed of fibrous tissue covered by the endocardium. At the base, they are continuous with one another and form a ring-shaped membrane around the margin of the arterial opening. Their pointed ends project into the ventricle and are attached by chordae tendineae, which attach to papillary muscles of the ventricle.

The **mitral valve** or **bicuspid valve** consists of two cusps which are connected in the same manner as the tricuspid valve except that it is much stronger and thicker in all parts. These valves permit free flow of blood from the atria to the ventricles but any backflow of blood causes these flaps to close.

The **semilunar valves** consist of three semilunar cusps which serve the same purpose as the mitral and tricuspid valves, in that they prevent backflow of blood. As each pocket fills with blood, they float out and distend until they meet and close off the vessel completely. These two semilunar valves are located between the right ventricle and the pulmonary artery, the pulmonary valve and between the left ventricle and the aorta, the **aortic valve**.



Major Blood Vessels

Inferior and Superior Vena Cava which carry deoxygenated blood from the body into the right atrium.

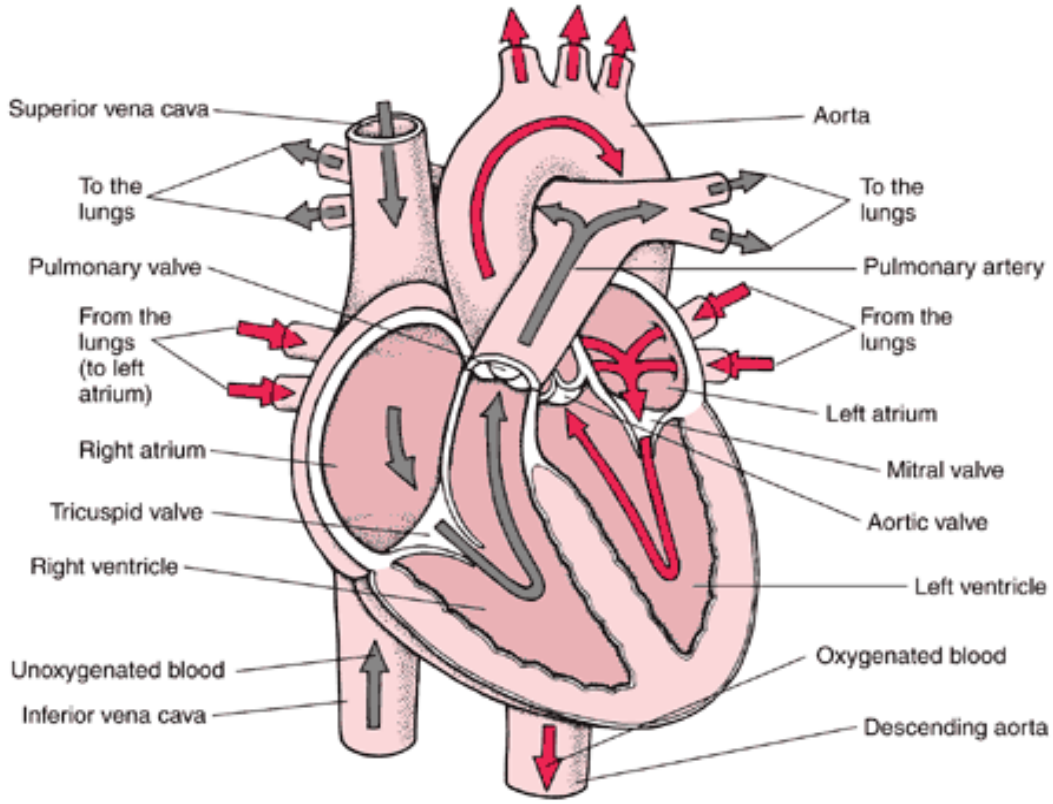
Pulmonary Artery which carries deoxygenated blood from the right ventricle to the lungs.

Pulmonary Veins which carry oxygenated blood from the lungs to the left atrium.

Aorta which carries oxygenated blood from the left ventricle to the body.

Pulmonary Versus Systemic Circulation	
• Deals with the right side of the heart	• Deals with the left side of the heart
• Deoxygenated blood enters the right side of the heart via the superior and inferior vena cava to the right atrium, passing through the tricuspid valve and into the right ventricle; blood exits through the pulmonary artery and subsequently the lungs	• Oxygenated blood enters the left side of the heart via the four pulmonary veins into the left atrium, passing through the mitral valve and into the left ventricle; blood exits through the aortic valve into the aorta and the remainder of the body
• Thin wall structure within the right ventricle due to projection of blood against low resistance	• Thick wall structure within left ventricle due to projection of blood against high resistance

***The pulmonary system also has a bronchial circulation which originates from the thoracic aorta and supplies oxygenated blood to the trachea, bronchi, nerves and the lung tissue itself.**



Coronary Arteries

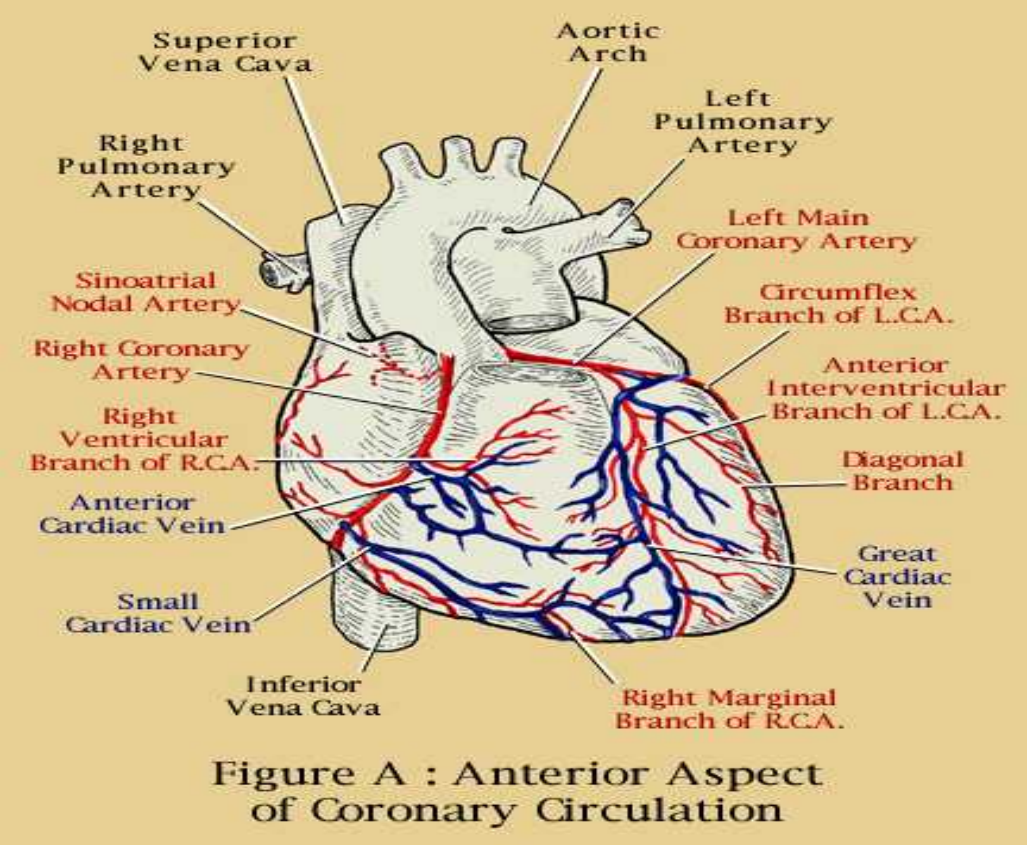


Figure A : Anterior Aspect of Coronary Circulation

Like any organ of the body, the heart has its own rich blood supply. The coronary arteries branch off from the base of the aorta and travel a considerable distance on the epicardial surface of the heart. They lie embedded in the fat that surrounds them and covers the heart. The two main coronary arteries are the right and left coronary arteries. They supply both the heart's electrical and mechanical structures.

Electrical = conduction system
Mechanical = heart muscle or myocardium

Right Coronary Artery

After leaving the aorta, the right coronary artery (RCA) passes diagonally toward the right side of the heart and descends in the groove between the right atrium and the right ventricle. Before passing around the posterior surface of the heart, it gives off its acute marginal branch which descends along the lateral side of the heart to the apex.

The right coronary artery supplies:

- 55% SA node
- 90% AV node
- a portion of the bundle of HIS
- posterior-inferior division of the left bundle branch
- posterior 1/3 of the septum
- right atrial and ventricular musculature
- inferior-posterior wall of the left ventricle

Left Coronary Artery

After leaving the aorta, the left coronary artery (LCA) passes behind the pulmonary artery and provides small branches to supply the left atrium. As it leaves the area of the pulmonary artery it branches off into two major divisions: LAD & LCX

Left Anterior Descending (LAD**)**

The left anterior descending (LAD) descends on the anterior surface of the heart in the groove between the right and left ventricles. It gives off many branches as it travels, with the largest being the diagonal branch which supplies the marginal wall of the left ventricle.

LAD supplies:

- anterior 2/3 of the septum
- the right bundle branch
- antero-superior division of the left bundle branch
- anterior wall of the ventricle

Circumflex

Circumflex artery (LCX) branches from the left coronary artery and passes to the posterior surface of the heart in the groove between the left atrium and left ventricle. It extends across the posterior of the heart. It has one major branch, the obtuse, which supplies most of the posterior surface of the left ventricle.

Left circumflex artery supplies:

- 45% of the SA node
- Infero-posterior division of the left bundle branch
- lateral wall of the left ventricle

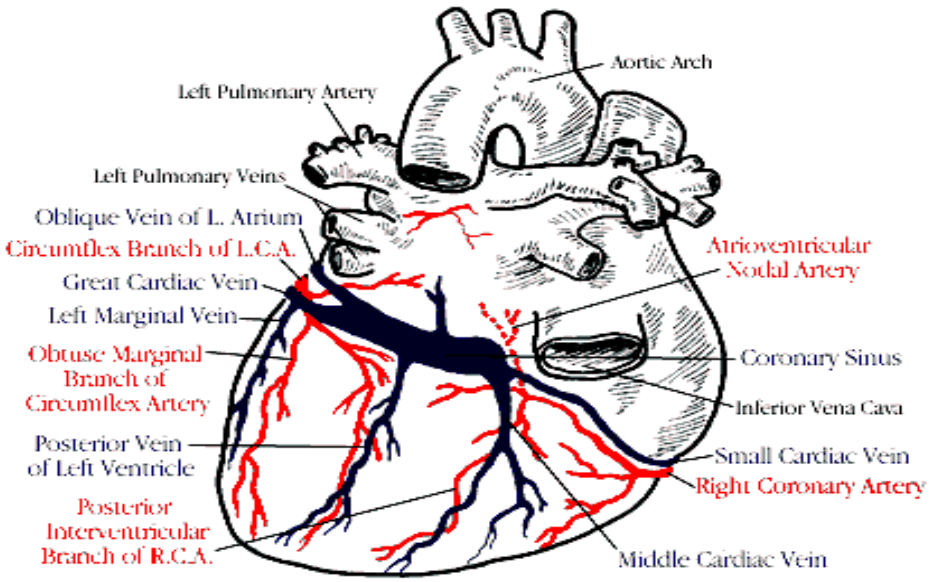
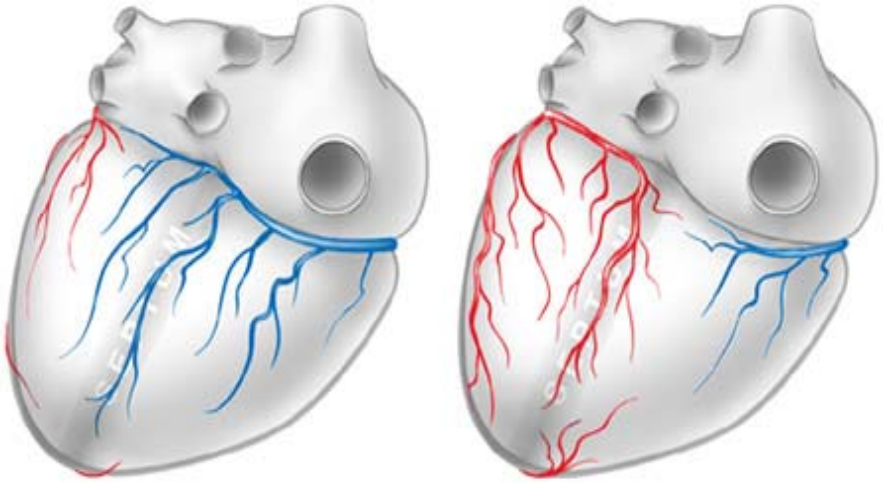


Figure B : Posterior Aspect of Coronary Circulation

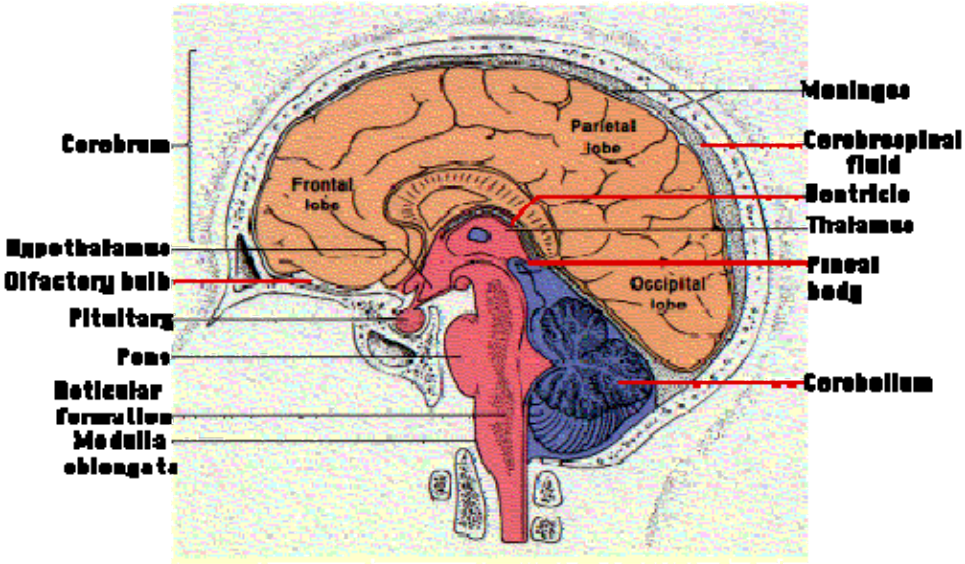


It is known that 80% of the population is ‘right dominant’; the posterior part of the heart is supplied by the RCA via the posterior branch called posterior descending artery (PDA).

Cardiac Veins

The venous drainage system closely parallels the arterial inflow, for example the great cardiac vein accompanies the LAD artery. All the veins flow into and from the coronary sinus which extends high across the posterior surface of the heart. The coronary sinus ends up in the right atrium.

Nervous Control of the Heart



Medulla

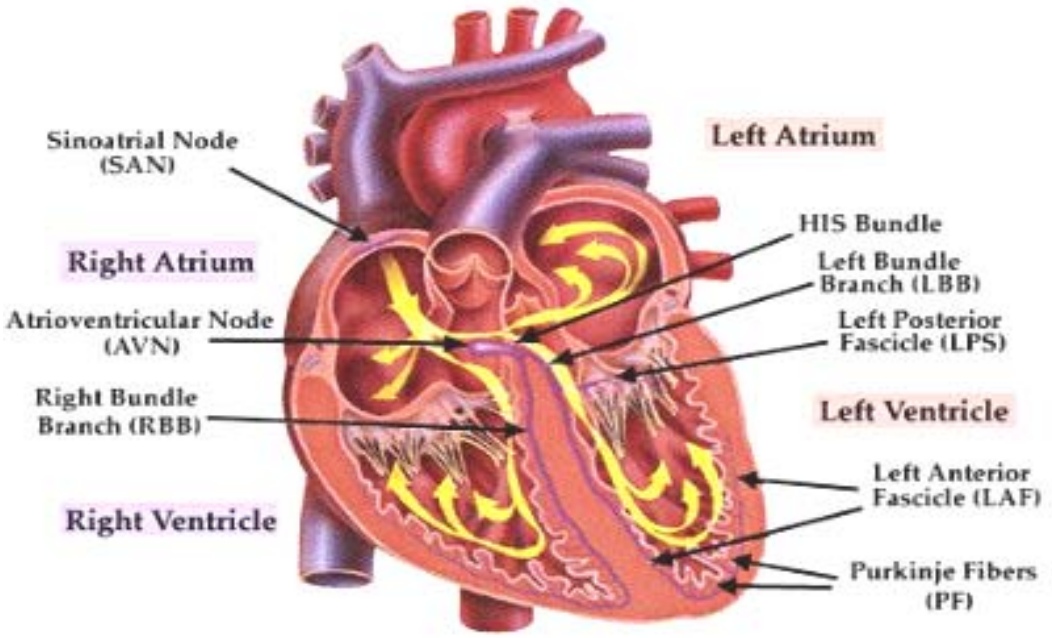
Central Nervous System

Autonomic Nervous System — Somatic Nervous System

Sympathetic Nervous System	**Parasympathetic Nervous System**
• mediator: norepinephrine	• mediator: acetylcholine
• Alpha receptors: found primarily in peripheral blood vessels stimulation > vasoconstriction blocking > vasodilatation	• only effect is a vagal response therefore it only has the ability to slow conduction
• Beta receptors: found primarily in the heart and lungs (Beta 1 indicates found only in the heart) stimulation - ↑ HR bronchodilation blocking - ↓ HR, bronchoconstriction	• Controls the SA node, atrial wall, junctional tissue but not the ventricles
• Controls the SA node, atrial wall, junctional tissue and ventricles.	

BOTH MAINTAIN A BALANCE!

Conduction System of the Heart



Electrical impulses within the heart are transmitted along a network of specialized cells called the conduction system. When the impulse reaches and stimulates the ventricular muscle, myocardial contraction occurs. Each normal heart beat is the result of an electrical impulse that originates in a specialized area in the wall of the right atrium called the SA node. This bundle of tissues acts as the battery for the heart and is the designated pacemaker. Other areas of the heart also have the ability to initiate impulses, but they assume this role only under abnormal circumstances.

Sinoatrial (SA node)

SA node is a specialized piece of tissue that can initiate its own impulse (property of automaticity). It is located in the posterior portion of the right atrium near the superior vena cava. It acts as the pacemaker of the heart. It initiates impulses at a rate of 60-100 per minute (Sinus rhythm).

Internodal Pathways

The internodal atrial conduction pathways conduct the electrical impulses rapidly from the SA node to the AV node.

Atrial-Ventricular (AV) Node

The AV node is located in the right atrium close to the septal leaflet of the tricuspid valve. Under normal conditions, the AV node is not usually the pacemaker, but it is capable of initiating impulses at a rate of 40-60 per minute (nodal or junction rhythm) if the SA node fails. The primary function of the AV node is to relay the electrical impulses from the atria into the ventricles in an orderly and timely way. A ring of fibrous tissue insulates the rest of the atria from the ventricles in order to prevent the electrical impulses from entering the ventricles haphazardly. The impulses travel relatively slowly through the AV node to reach the **bundle of His**. This allows the atria to contract and empty and the ventricles to fill before the ventricles contract.

Bundle of His

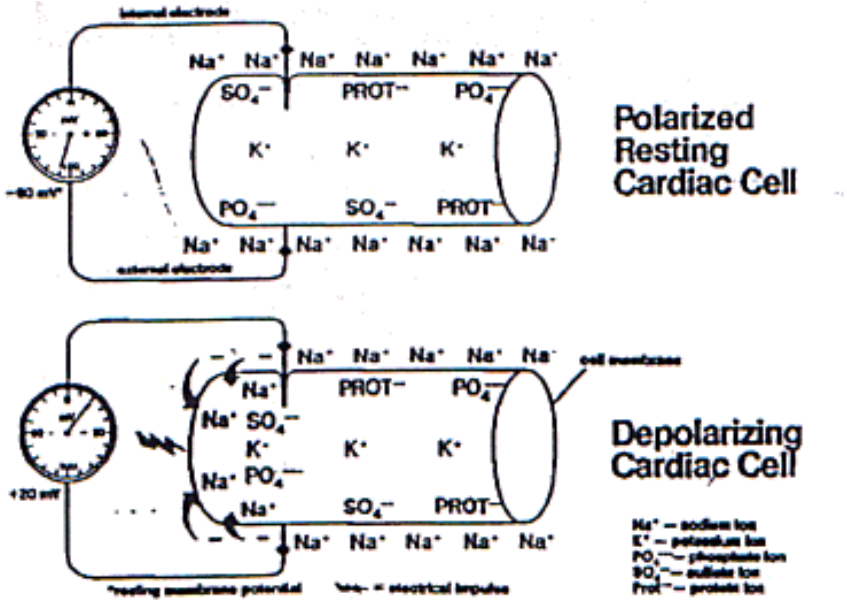
This bundle lies in the upper part of the interventricular septum, connecting the AV node with the two bundle branches. Once the electrical impulses enter the bundle of His, they travel more rapidly to the bundle branches.

Right and Left Bundle Branches and Purkinje Fibers

The right and left bundle branches arise from the bundle of His, straddle the interventricular septum and travel down either side of the septum. They subdivide into smaller and smaller branches and connect with the Purkinje network. The ventricular conduction tissue, an intricate web of Purkinje fibers, is spread diffusely throughout the myocardium ending at the muscle fibers. This network also has the ability to initiate impulses at a rate of 15-40 per minute (ventricular rhythm).

Electrophysiology of the Heart

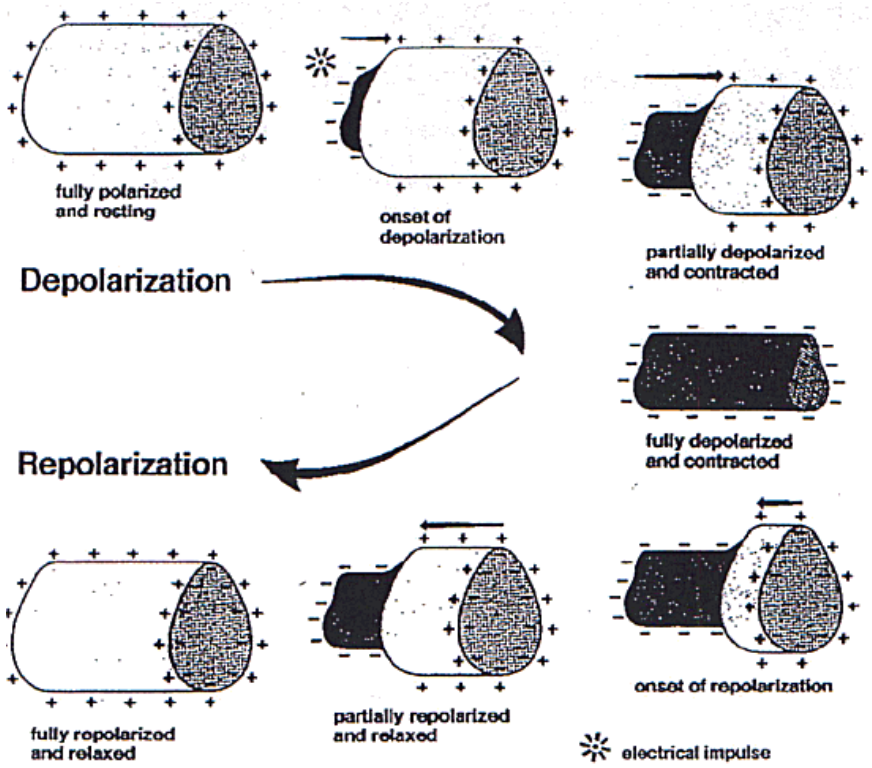
Cardiac cells are able to generate and conduct electrical impulses which are responsible for the contraction and relaxation of myocardial cells. These electrical impulses are the result of brief but rapid flow of positively charged ions back and forth across the cardiac cell membrane. The difference in the concentration of these ions across the cell membrane at any given time is called the **electrical potential** and is measured in millivolts.



Resting State

When the cardiac cell is in the resting state, a high concentration of positively charged sodium ions (Na^+) is present *outside* the cell. At the same time, a high concentration of both positively charged potassium (K^+) ions and negatively charged ions (organic phosphate ions, organic sulfate ions, protein ions) is present *inside* the cell. When the interior of the cell is electrically negative in reference to its positive exterior, a **negative electrical potential** exists across the cell membrane. This is possible because the cell membrane is impermeable to positively charged sodium ions during the resting state and negatively charged phosphate, sulfate and protein ions at all times. When a cell membrane is impermeable to an ion, it does not allow the free flow of that ion across it.

When the ions are aligned with a layer of positive ions surrounding the cell membrane and an equal number of negative ions lining the inside of the cell membrane opposite to each positive ion, the resting cell is **polarized**. The electrical potential across the membrane of a resting cardiac cell is called the **resting membrane potential**. The resting membrane potential in myocardial cells (atrial and ventricular) and the electrical conduction system is normally –90mV (except SA and AV nodes, usually –70mV).



Depolarization and Repolarization

When stimulated by an electrical impulse, the polarized myocardial cell membrane becomes permeable to positively charged Na^+ ions, allowing Na^+ to flow into the cell causing the interior of the cell to become less negative. When the resting membrane potential drops from –90mV to about –60mV, large pores in the membrane open momentarily. These pores are called the **fast sodium channels** which allow the rapid, free flow of Na^+ across the cell membrane. This results in a sudden large influx of positively charged Na^+ ions into the cell causing the exterior of the cell to become rapidly negative with respect to the now positive interior. This reversal of the cell's resting, polarized state is called **depolarization**.

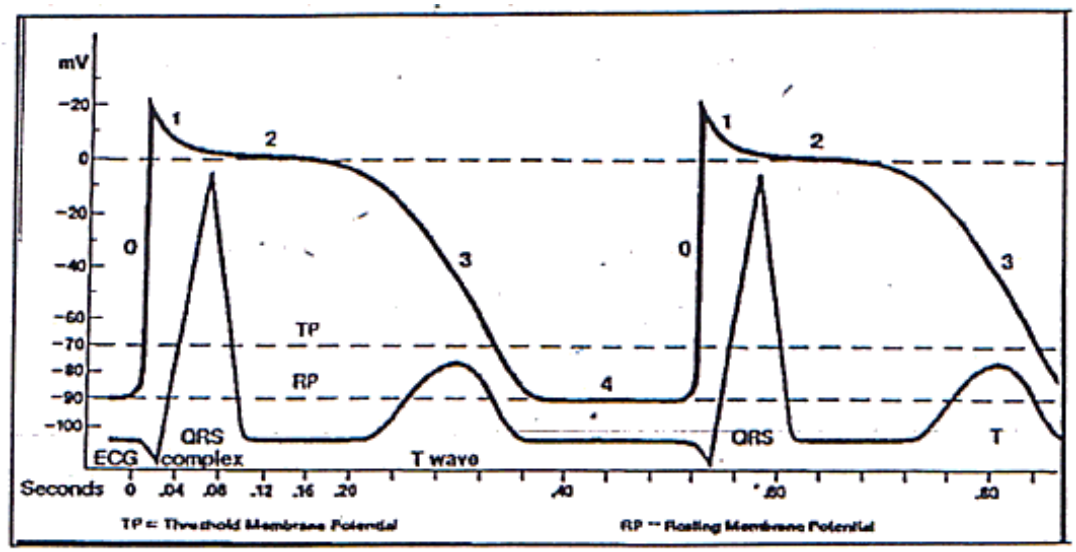
The fast sodium channels are usually found in the myocardial cells and the cells of the electrical conduction system (except SA and AV nodes). The cells of the SA and AV nodes have **slow calcium-sodium channels** which open when the membrane potential drops to about –50mV. They allow the entry of positively charged Ca^{++} and Na^+ ions into the cells during depolarization at a slow and gradual rate. The result is a slower rate of depolarization as compared to the depolarization of cardiac cells with fast sodium channels.

As soon as a cardiac cell depolarizes, positively charged potassium ions flow out of the cell, initiating the process by which the cell returns to its resting polarized state. This repolarization process involves a complex exchange of Na^+, Ca^{++} and K^+ ions across the cell membrane.

Depolarization of one cardiac cell acts as an electrical impulse on adjacent cells and causes them to depolarize. An electric current is produced in the direction of depolarization as the electrical impulse flows from cell to cell. As the cells repolarize, another electric current is produced. The direction of flow and magnitude of currents initiated by depolarization and repolarization can be detected by surfaced electrodes and recorded as the electrocardiogram. (Huszar, 1988)

Threshold Potential

A cell does not need to be completely repolarized to its resting polarized state before it can be stimulated to depolarize again. The cells of the SA and AV nodes can be depolarized when they have been repolarized to about –30 to –40 mV. The rest of the cells of the electrical conduction system and myocardial cells can be depolarized when they have been repolarized to about –60 to –70mV. The level to which a cell must be repolarized before it can be depolarized again is called the **threshold potential**. A cardiac cell cannot be stimulated to generate or conduct an electrical impulse or to contract until it has been repolarized to a threshold potential. (Huszar, 1988).



Cardiac Action Potential

A cardiac action potential is a schematic representation of the changes in the membrane potential of a cardiac cell during depolarization and repolarization. It is divided into five phases:

Phase 0 – Depolarization Phase

Phase 0 is the tall upstroke of the action potential during which the cell membrane reaches the threshold potential, triggering the fast sodium channels to momentarily open and allow the rapid entry of sodium into the cell. As the positively charged ions flow into the cell, the interior of the cell becomes electrically positive to about +20mV with respect to its exterior. During the upstroke, the cell depolarizes and begins to contract.

Phase 1 – Early Rapid Repolarization Phase

During Phase 1, the fast sodium channels close, stopping the flow of sodium into the cell, followed by the loss of potassium from the cell. The end result is a decrease in the number of positive electrical charges within the cell and a drop in the membrane potential to about 0mV.

Phase 2 – Plateau Phase

This is the prolonged phase of slow repolarization of the action potential of the myocardial cell, allowing it to finish contracting and begin relaxing. The membrane potential remains about 0mV because of the very slow rate of repolarization. The complicated exchange of ions across the cell membrane occurs; calcium slowly enters the cell through the slow calcium channels as potassium continues to leave the cell and sodium to enter it.

Phase 3 – Period Between Action Potentials

During Phase 3, the inside of the cell becomes markedly negative and the membrane potential returns to about –90mV. This is primarily due to the flow of potassium from the cell. Repolarization is complete by the end of Phase 3.

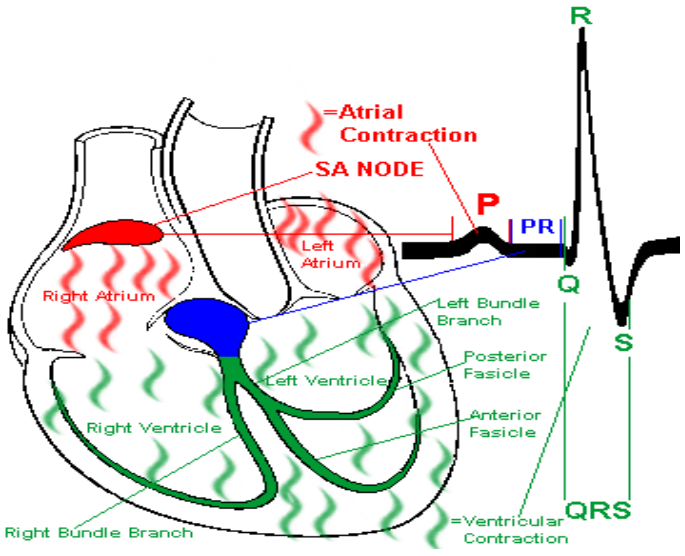
Phase 4 – Period Between Action Potentials

The membrane has returned to its resting potential and the inside of the cell is again negative at –90mV with respect to the outside. However, there is still an excess of sodium in the cell and an excess of potassium outside the cell. A mechanism known as the **sodium-potassium pump** is activated at this point. This mechanism transports excess sodium out of the cell and potassium back in. (Huszar, 1988)

Refractory and Supernormal Periods

The refractory period of cardiac cells extends from Phase 0 to the end of Phase 3 of the cardiac action potential (beginning with the onset of the QRS complex and ending with the end of the T wave). It is divided into the absolute and relative refractory periods.

Absolute refractory period:	• ends midway through Phase 3 (corresponds to about the peak of the T wave) • cardiac cells have not repolarized to their threshold potential and cannot be stimulated to depolarize • myocardial cells cannot contract and cells of the electrical conduction system cannot conduct an impulse
Relative refractory period:	• extends through the second half of Phase 3 (corresponds to the downslope of the T wave) • cardiac cells have repolarized to their threshold potential and can be stimulated to depolarize if the stimulus is *strong* enough
Supernormal period:	• during a short portion of Phase 3, just before the cells return to their resting potential, a stimulus weaker than normally required can depolarize cardiac cells (Huszar, 1988) • also known as the vulnerable period of repolarization



Excitability and Automaticity

Property of excitability	• the capability of a resting, polarized cardiac cell to depolarize in response to an electrical stimulus • all cardiac cells have this property
Property of automaticity	• the capability of a cardiac cell to depolarize spontaneously during Phase 4 (to reach threshold potential and to depolarize completely without being externally stimulated)

Spontaneous depolarization depends on the ability of the cell membrane to become permeable to sodium during Phase 4, allowing a steady leakage of sodium ions into the cell. This causes the resting membrane potential to become progressively less negative. When the threshold potential is reached, rapid depolarization occurs.

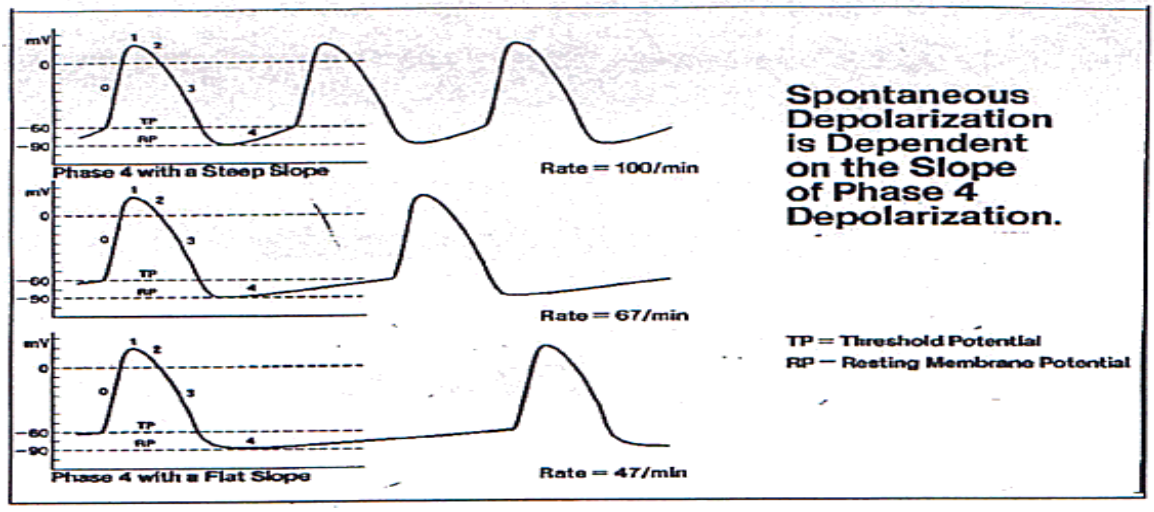
The **rate of spontaneous depolarization** is dependant on the **slope of Phase 4 depolarization.** The **steeper** the slope, the **faster** the rate of spontaneous depolarization and the **rate of impulse formation (firing rate)**,the **flatter** the slope, the slower the firing rate.

Automaticity - normally common to the pacemaker cells in the:
- SA node
- some parts of the internodal atrial conduction tracts
- AV node
- all parts of the bundle of His, bundle branches and Purkinje fibers

The pacemaker cells other than those in the SA node hold this property in reserve if the SA node should fail, thus they are called **latent pacemaker cells**.

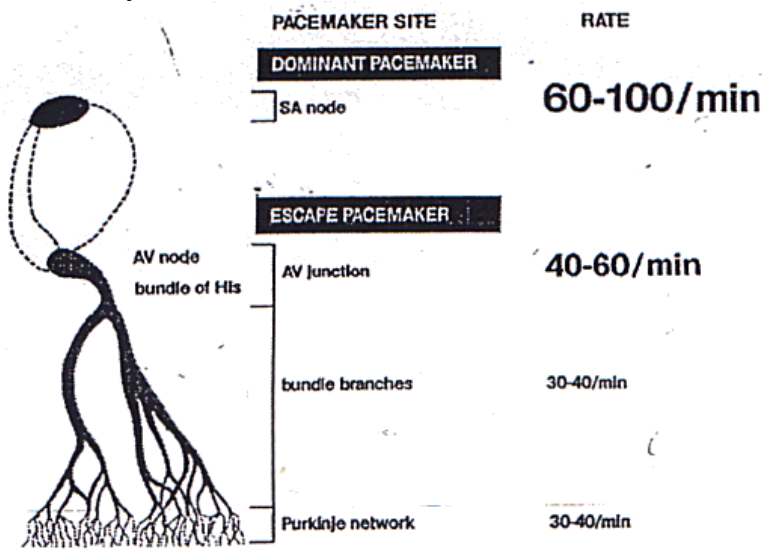
Myocardial cells, which do not normally have the capability to depolarize spontaneously during Phase 4, are called **nonpacemaker cells**.

↑ sympathetic activity, administration of catecholamines	▸ ↑ slope of Phase 4 depolarization ↑ in automaticity and firing rate
↑ parasympathetic activity, administration of lidocaine, procainamide, quinidine	▸ ↓ slope of Phase 4 depolarization ↓ automaticity and firing rate (Huszar, 1988)



Overdrive Suppression

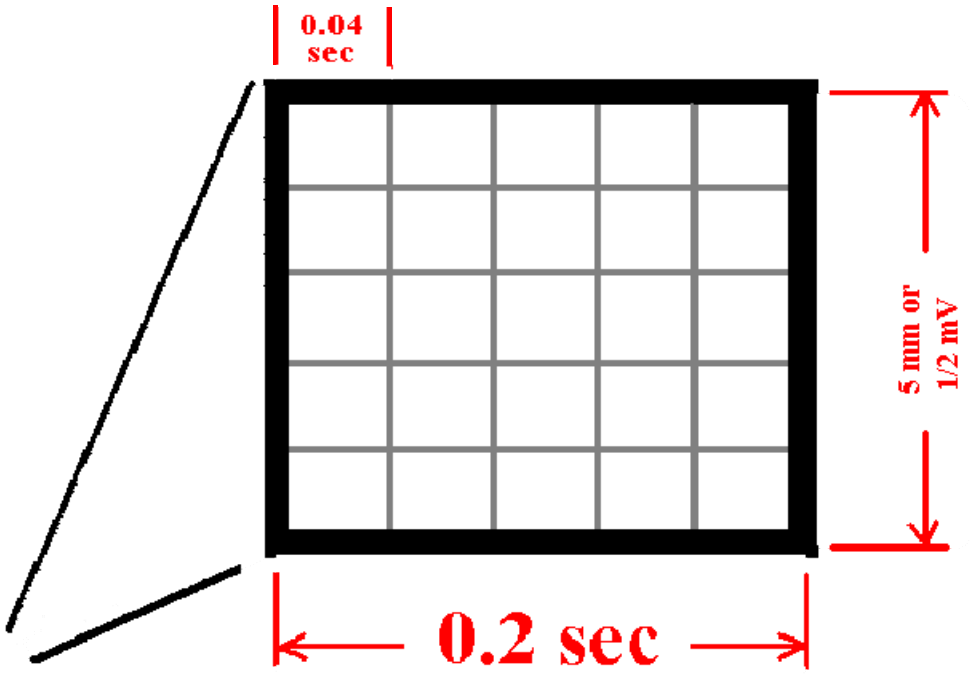
Usually the pacemaker cells with the fastest firing rate control the heart rate at any given time. Each time these pacemaker cells generate an electrical impulse, the slower firing pacemaker cells are depolarized before they can fire spontaneously.



The Electrocardiogram

The electrocardiogram (ECG or EKG) 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.

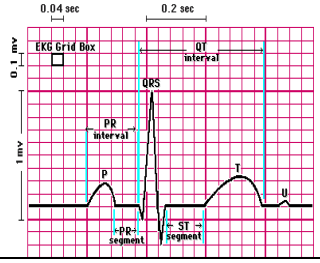
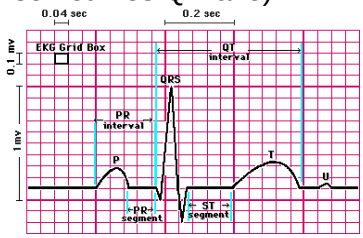
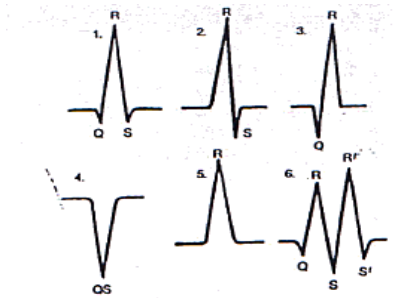
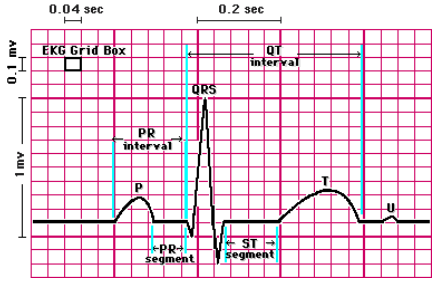
ECG Paper

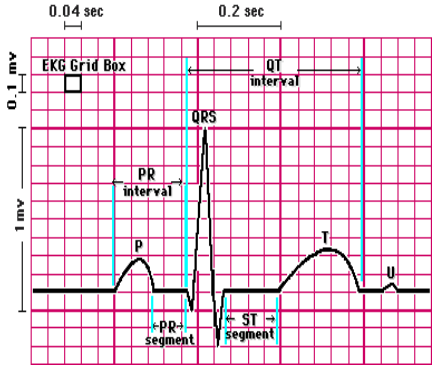
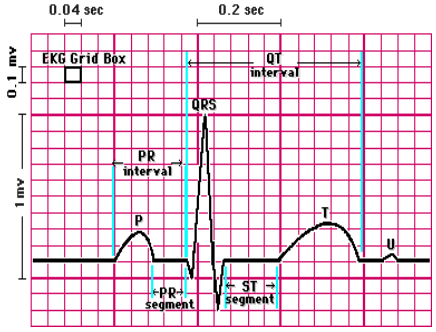
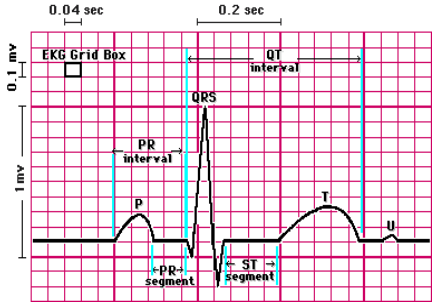
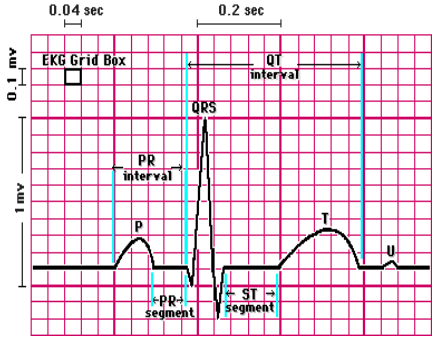


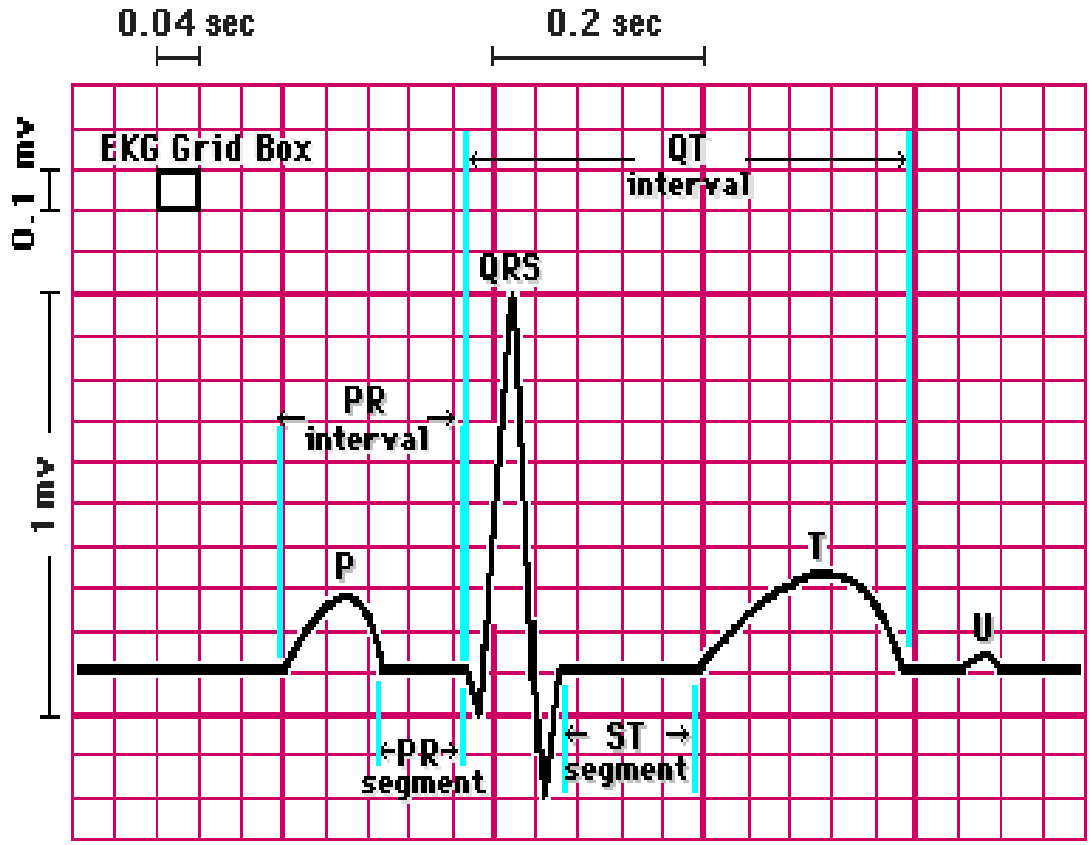
- The measurement of **time** is calculated along the horizontal lines in seconds.
  Each small square = .04 sec.
  Each large square = .20 sec.

- The voltage of **energy** is represented along the vertical lines in millivolts or millimeters.
  Each small square = .1 mV or 1 mm
  Each large square = .5 mV or 5 mm

Components of the Electrocardiogram

ECG Component	Location	Normal Values	Electrical Activity
P Wave 	**Measured** from the beginning of the P wave to the end of the P wave	.1 to .3 mV	**Represents** atrial depolarization
<u>QRS Complex</u> **Q wave** – the first negative deflection following the P wave (not preceded by an R wave) **R wave** – the first positive deflection extending above baseline following the P wave (and Q wave if present) **S wave** – the first negative deflection extending below baseline following the R wave (or sometimes Q wave)  	**Measured** from the point where the first wave in the complex begins to deviate (abruptly or gradually) from the baseline to the point where the last wave of the complex begins to flatten out (abruptly or gradually) at, above or below the baseline. The QRS complex may be composed of all 3 waves (QRS) or 2 of the 3 (QR or RS) or simply an R wave.	0.04 to .12 sec Q wave – significant if *wider than .04 sec *greater than 1/3 of height of the R wave	**Represents** depolarization of the right and left ventricles.
T wave 	**Measured** from the onset of the T wave to the end of the T wave.	.10 to .25 sec.	**Represents** ventricular repolarization
ECG Component	**Location**	**Normal Values**	**Electrical Activity**

PR Interval 	**Measured** from the onset of the P wave to the onset of the QRS complex (the first positive or negative deflection from the isoelectric line following the P wave)	0.12 to .20 sec	**Represents** the time of progression of the electrical impulse from the SA node through the entire electrical conduction system to the ventricular myocardium
ST Segment 	**Measured** from the end of the QRS complex to the onset of the T wave	Normally flat	**Represents** the early part of repolarization of the right and left ventricles
QT Interval 	**Measured** from the beginning of the Q RS complex to the end of the T wave	0.32 to 0.40 sec (rate related)	**Represents** the total ventricular activities; depolarization and repolarization
U wave 	**Measured** from the beginning of the U wave to the end of the U wave	Amplitude is usually < 2 mm and always smaller than the preceding T wave	**Probably represents** the final stage of repolarization of the ventricles


0.04 sec
0.2 sec
EKG Grid Box
0.1 mv
1 mv
QT
interval
QRS
PR
interval
P
T
U
PR
segment
ST
segment

ECG Rhythm Analysis

1. Rhythm/Regularity

- Is the distance between each R-R wave constant? If so, the rhythm is considered regular.
- Does the distance between each R-R wave vary? If the difference is greater than .12 seconds, the rhythm is considered irregular or abnormal.

2. Rate

- Calculate the heart rate:
    i. **If rhythm is irregular** - Count the number of R waves in a 6 second strip, then multiply by 10. This calculation gives an approximate rate.

    ii. **If the rhythm is regular** - Count the number of large squares (assign a value of 1.0 to each large square) and the number of small squares (assign a value of .20 to each small square) between 2 R waves or P waves. Divide this value into 300 (the number of large squares in one minute). This will give an accurate heart rate.

- Is the calculated rate within the accepted norm of 60-100 per minute? A value greater than 100 is considered a ***tachycardia*** and a value less than 60 is considered a ***bradycardia*****.**

3. P Waves

- Is there a P wave preceding each QRS complex?
- Is the P-P distance constant or does it vary?
- What is the shape of each P wave? Is the shape constant? Are they smoothly rounded and upright, biphasic or inverted?
- What is the site of origin of the electrical impulse? Is it the SA node, AV node or the junction tissue? i.e. if P waves are present and upright before each QRS complex, the SA node is the site of origin.

4. PR Interval

- Does the PR interval fall within the normal range of .12 to .20 seconds?
- A delay beyond .20 seconds indicates a delay in the conduction from the SA node to the ventricles.
- A PR interval of less than .12 seconds indicates the impulse has reached the ventricles through an abnormal pathway.

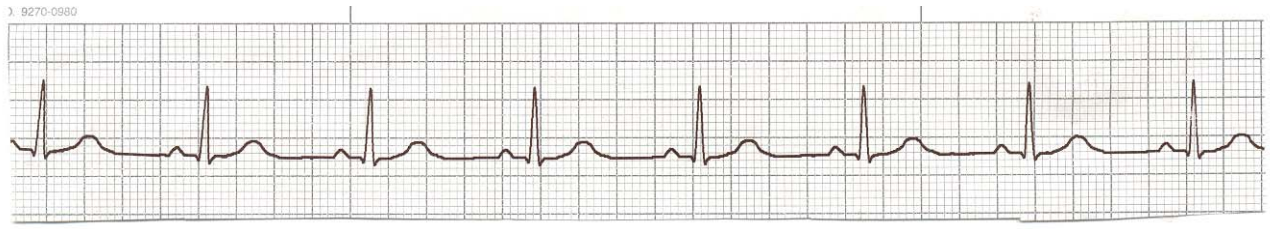
5. QRS Complexes

- Is there a QRS complex following each P wave?
- Is the QRS complex of normal duration, .04 to .12 seconds?
- A widened complex indicates an obstruction in one of the bundle branches and a delay in activation of the ventricles.

Therefore, **NORMAL SINUS RHYTHM** consists of:

- Rate – 60 to 100/min. (ventricular)
- Rhythm – Regular
- P waves – Present, upright(or biphasic or inverted in V_1) before QRS complexes, rate 60 - 100/min
- PR Interval - .12 to .20 sec
- QRS Complex - .04 to .12 sec

Example of NSR



Rate –71/min. 300 ÷ 4.2=71)
Rhythm – R waves regular with no variation greater than .0.12 seconds
P waves – present and upright before each QRS complex; 1:1 conduction
PR Interval - .20 sec. (normal)
QRS Complex - .06. (normal); morphology: qRs
Interpretation – Normal Sinus Rhythm

Sign of Ischemia – ST segment depression

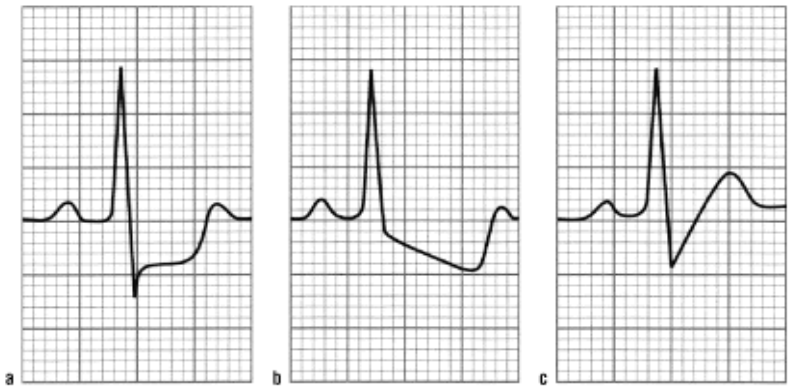
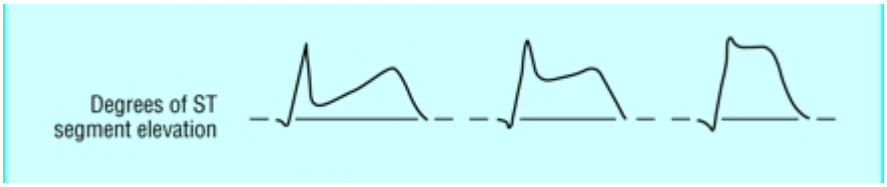
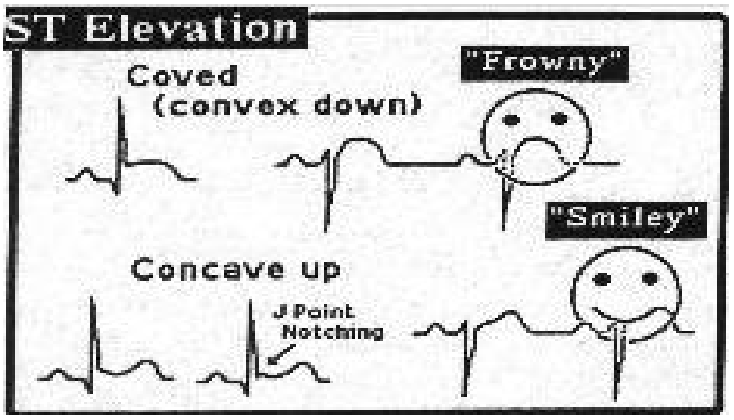


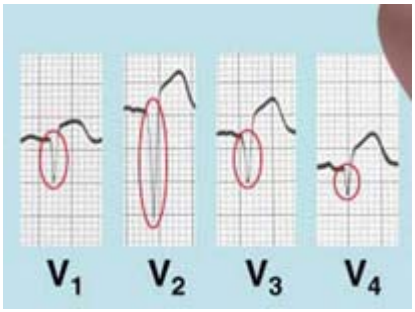
Figure 1. Various forms of ST-segment depression during exercise stress testing. a. Horizontal. b. Downsloping. c. Upsloping. Horizontal and downsloping forms indicate ischemia; upsloping is a poor indicator of ischemia.

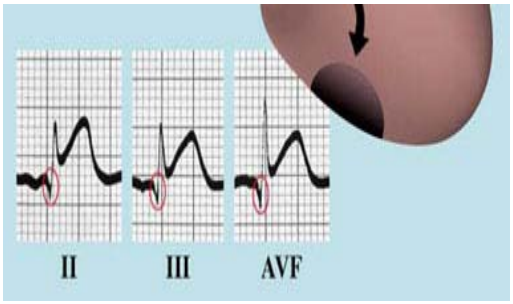
Sign of Injury – ST segment elevation





Sign of Infarction (necrosis) – Abnormal Q-wave



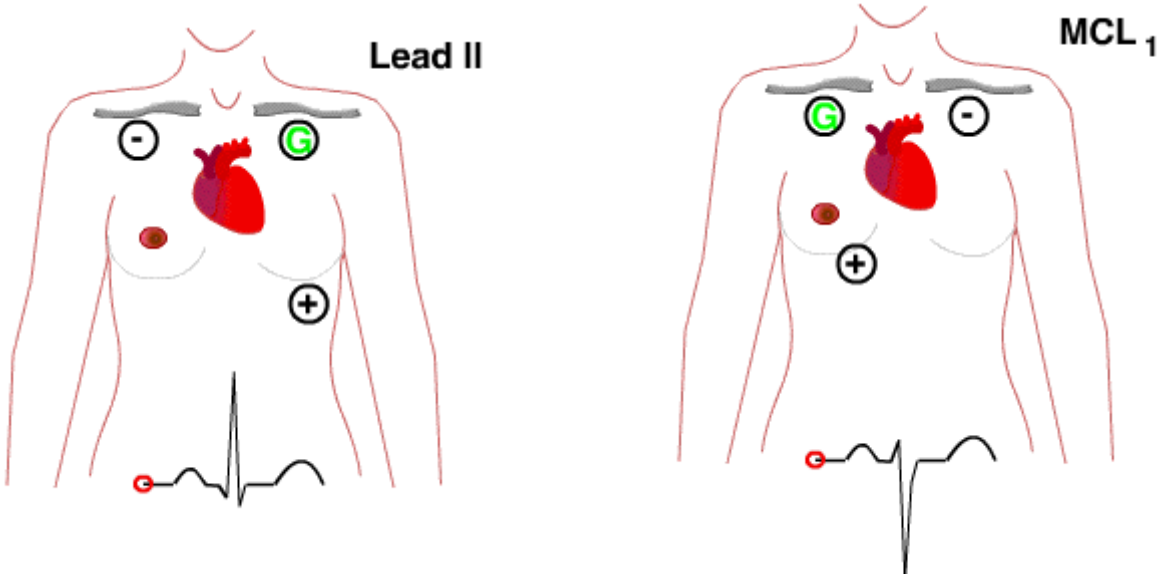


Monitoring Leads

Electrical flow in the heart is measured by externally applying electrodes in relationship to a direct line, called an **axis**, between the two poles. A 3 lead system consists of one negative pole, one positive pole and one groundThere are 12 established leads, each of which has a unique individual axis. Any lead may be used to continuously monitor cardiac activity. Some leads are more popular for monitoring than others.

Limb lead II and **chest lead V_1** or **modified chest Lead I** (**MCL_1**) are the two most commonly used monitoring leads. Limb lead II is the most traditional monitoring lead but has the disadvantage of being in the way if defibrillation is required. **Chest lead V_1 or MCL_1** does not interfere with defibrillation or auscultation but produces an ECG complex slightly different from the traditional lead II complex. (Wiederhold, 1988). Clinically, chest lead V1 or MCL1 is more diagnostic and informative

3 electrode placement = Lead 1, II, III or MCL_1



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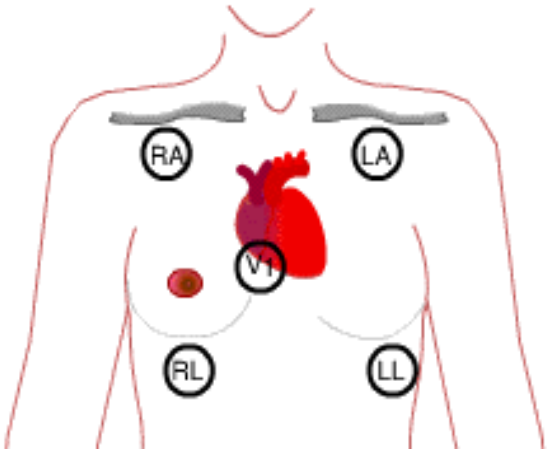
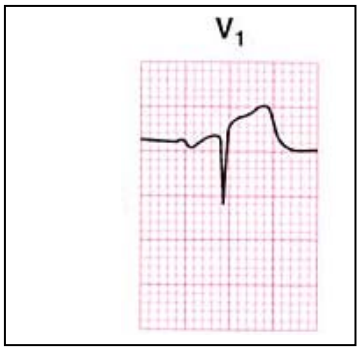
Advantages for the Use of MCL_1 or V_1 Monitoring

- Identifies atrial and ventricular arrhythmias
- Identifies bundle branch blocks: LBBB and RBBB
- Provides more accurate information in the ventricular conduction system.
    - Early recognition of developing right bundle branch block in patients with anteroseptal or extensive anterior wall MI
    - Identification of aberrantly conducted premature beats or PVCs originating from the right or left ventricle.
    - With pacemakers, QRS configuration identifies the area of lead placement (which should be the right ventricle).
- Provides assessment of the posterior view of the heart

5 electrode placement = Standard limb leads/views (Leads 1. II, III, aVR, avL, avF) + V_1

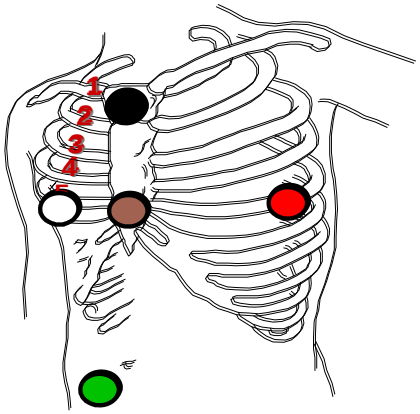




Normal morphology for V1 is a biphasic P wave , small r wave and large S wave

EASI lead placement = able to monitor any views or leads + derived 12-Lead ECG

EASI Lead Placement



Adult/ Pediatric Patient

E- Brown electrode– lower sternum at 5th ICS

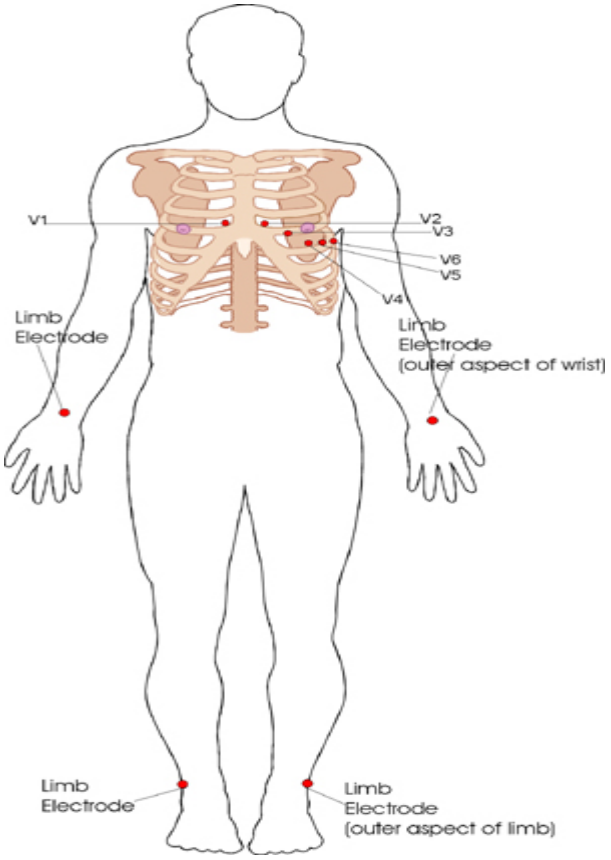
A- Red electrode- left midaxillary line at the same level as the E electrode (5th ICS)

S- Black electrode- on the upper sternum

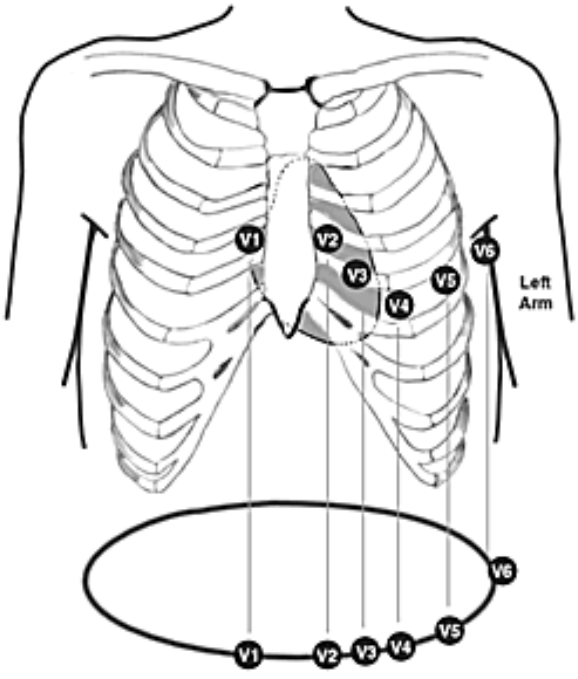
I- White electrode- right midaxillary line at the same level as the E electrode (5th ICS)

Reference- Green electrode- ground electrode usually below the 6th rib

10 Electrode Placement = 12 Leads or 12 views (Standard 12-Lead ECG)



Precordial (chest) lead placement = V_1, V_2, V_3, V_4, V_5, V_6 or horizontal view of the heart



Lead V_1 **The electrode is at the fourth intercostal space just to the right of the sternum. (4th ICS RSB)**

Lead V_2 The electrode is at the fourth intercostal space just to the left of the sternum. **(4th ICS LSB)**

Lead V_3 The electrode is at the line midway between leads V_2 and V_4.

Lead V_4 The electrode is at the midclavicular line in the fifth intercostal space. **(5th ICS MCL)**

Lead V_5 The electrode is at the anterior axillary line at the same level as lead V_4. **(5th ICS AAL)**

Lead V_6 The electrode is at the midaxillary line at the same level as lead V_4. **(5th ICS MAL)**

NOTE: The characteristics of normal sinus rhythm remain the same no matter which lead is chosen for monitoring.

Laws of Electrocardiography

There are 3 basic laws of electrocardiography which indicate how the propagated action potential wave (the cardiac conduction cycle) is interpreted by the electrodes of any lead.

1. A positive deviation will be recorded in any lead if the wave of depolarization spreads toward the positive electrode on the axis of the lead.

2. A negative deviation will be recorded in any lead if the wave of depolarization spreads toward the negative electrode on the axis of the lead.

3. A biphasic (both positive and negative) deflection will be recorded in any lead if the wave of depolarization spreads away from the axis of the lead. (Wiederhold, 1988)

INTERPRETATION OF HEART RYTHMS AND RELATED TREATMENTS

Initial Steps in the Assessment of Cardiac Arrhythmias

1. Early recognition of changes in patient's rhythm.
    - Awareness of contributing factors or causes of arrhythmias i.e. electrolyte imbalances (potassium, calcium and magnesium in particular), hypoxemia, myocardial ischemia, injury and infarction, acidosis, drug induced (prolonged QT interval), and anemia.

2. Immediate assessment of patient's condition to determine degree of hemodynamic compromise, if any.

    This includes:
    - Level of consciousness
    - Cardiac output –assessed by systemic blood pressure and presence of palpable pulses.

3. Notifying physician of change in patient's condition.

4. Accurate recording of rhythm strips and patient assessment.

5. Appropriate treatment if required (initiation of CPR, the use of appropriate life-saving drugs i.e. Atropine, Amiodarone, and Epinephrine, and application of electrical therapy i.e. defibrillation and application of TCP).

6. Assessment / evaluation of treatment.

NOTE: Depending on the type of arrhythmia, treatment may be initiated prior to notifying physician.

Arrhythmias Originating in the Sino-atrial Node (SA node)

This group of arrhythmias results from disturbances of impulse formation in the SA node.

The SA node retains its normal roles as pacemaker, but instead of discharging impulses at regular intervals 60 – 100 times per minute, the rate exceeds 100 per minute or less than 60 per minute, or the node does not discharge rhythmically. In some instances the pacemaker site wanders from the SA node to nearby areas. As a general rule, sinus node disorders are not dangerous and can be considered as minor arrhythmias. However, if the heart rate is very slow (less than 50 per minute) or very fast (greater than 130 per minute) the risk is significantly increased, in which case the arrhythmia would be classified as a major arrhythmia.

1) Sinus Tachycardia

Mechanism
The SA node is the pacemaker and discharges impulses regularly at a rate faster than 100 per minute. This increase in heart rate is usually due to overactivity of the sympathetic nervous system, which usually results from some additional stressor, i.e. fever, anxiety, physical stress, or "heart failure".

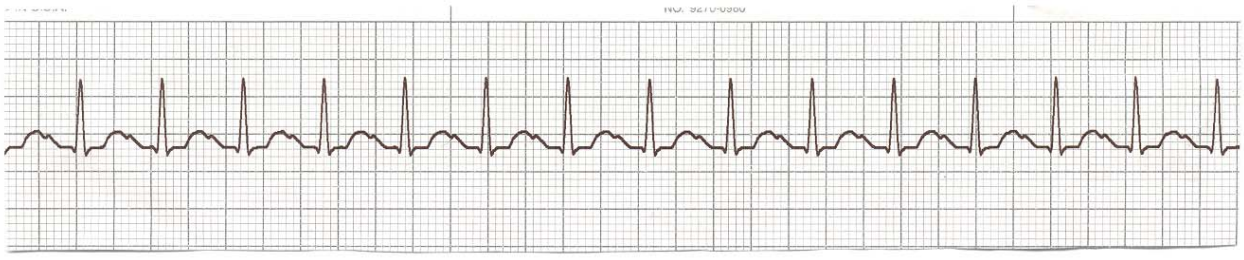
Characteristic features/analysis
- **Rate** – Usually 100 – 150 beats per minute
- **Rhythm** – Regular
- **P waves** – Normal and precede each QRS complex
- **PR Interval** - Normal
- **QRS Complex –** Normal

Hemodynamic effect
- Usually produces no symptoms especially low tachycardia rate of 101 -120/minute
- May produce cardiac or chest pain due to:
  - shortened diastolic time resulting in reduced coronary perfusion time
  - shortened filling time resulting in reduced stroke volume (cardiac output)
  - increased workload resulting in increased oxygen demand

Treatment
The initial step in treating sinus tachycardia is to identify the underlying cause rather than slowing the rate. If possible, treatment should be directed at treating the basic problem i.e. if hypovolemia - infuse volume or increase fluid intake, if febrile - decrease temperature, if anxious – administer anti-anxiety drugs as ordered.

Pharmacologic therapy: for coronary artery disease management, beta blocking agents, calcium channel blockers or digoxin are ordered to reduce heart rate.

Example of Sinus Tachycardia



Rate –136/minute
Rhythm – Regular with 1:1 conduction
P wave – Normal
PR Interval – 0.16 seconds
QRS Complex – 0.08 seconds, Morphology: qRs
Interpretation – Sinus Tachycardia

2) Sinus Bradycardia

Mechanism
Sinus bradycardia exists when the SA node discharges regularly at a rate slower than 60 beats per minute. The underlying cause is usually due to a parasympathetic (vagal) dominance of the SA node.

NOTE: Sinus bradycardia occurs more frequently with inferior wall myocardial infarctions because of the ischemia to the SA node.

Characteristic features/analysis
- **Rate** – 40-60 beats per minute
- **Rhythm** – Regular
- **P waves** – Normal and precede each QRS complex
- **PR Interval** - Normal
- **QRS Complex –** Normal

Hemodynamic effect
- Reduced cardiac output due to slow rate

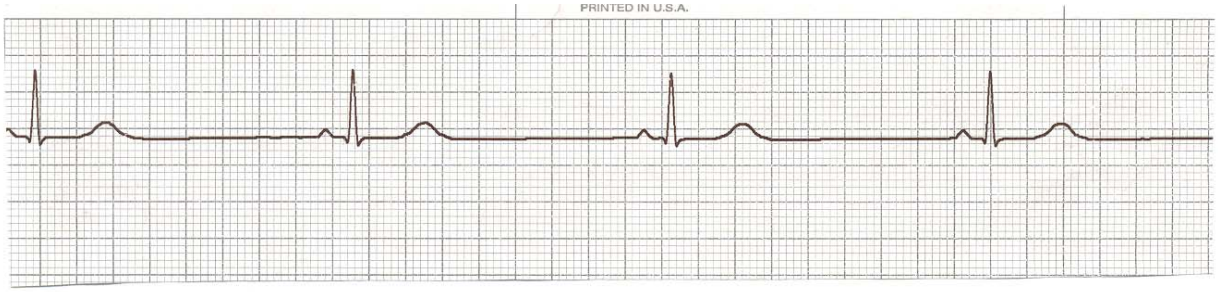
Treatment
Sinus bradycardia should be treated only if one of the following occur:
- reduction in cardiac output
- PVC's develop
- Rate remains less than 50 beats per minute

Nursing intervention: Assess patient's medication profile to identify possible drugs with known bradycardia effects

Pharmacologic treatment includes: Atropine and Dopamine or Epinephrine infusion

Electrical therapy: application of TCP or insertion of temporary transvenous pacemaker

Example of Sinus Bradycardia



Rate –36 beats/minute
Rhythm – Regular; 1:1 conduction
P wave – Normal
PR Interval – 0.20 seconds
QRS Complex – 0.08 seconds; morphology: qRs

3) Sinus Arrhythmia

Mechanism
The impulse arises from the SA node but not with a completely regular rhythm.

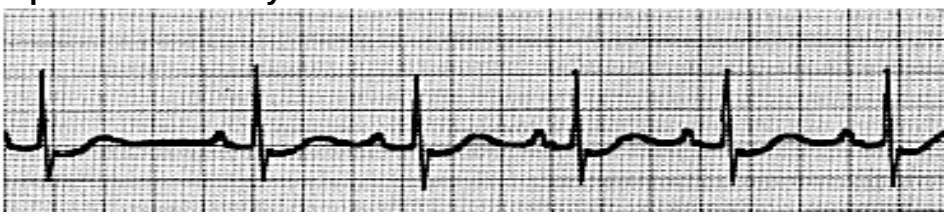
The irregularity is due to variation of vagal influence on the SA node which results in "alternating" periods of slow and fast rates. Usually this is related to the phase of respiration (rate increases during inspiration and slows during expiration). This rhythm is commonly observed in children.

Characteristic features/analysis
- **Rate** – 50-60 beats per minute
- **Rhythm** – irregular
- **P waves** – Normal and precede each QRS complex
- **PR Interval** - Normal
- **QRS Complex –** Normal

Hemodynamic effect
None

Treatment
No treatment is necessary.

Example of Sinus Arrhythmia



Rate –70-100 beats/minute
Rhythm – Irregular
P wave – Normal
PR Interval – 0.20
QRS Complex – 0.06 seconds; morphology: Rs

4) Wandering Pacemaker

Mechanism
Impulses arise in a normal manner in the SA node but the pacemaker wanders from the SA node to the atria and/or the AV nodal area.

This is demonstrated on the ECG by transient changes in the size, shape and direction of the P waves. As with sinus arrhythmia, wandering pacemaker is caused by varying vagal tone.

An increased vagal tone caused the SA node to slow, thus allowing adjacent tissue to take over as pacemaker temporarily. As vagal tone decreases, the rate of SA node impulses increases and the SA node becomes the dominant pacemaker again.

Characteristic features/analysis

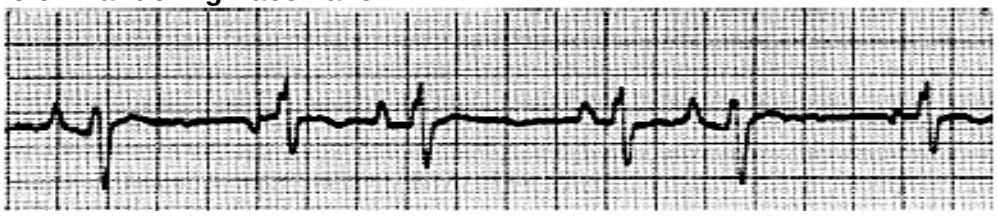
- **Rate** – usually normal (maybe slow)
- **Rhythm** – irregular
- **P waves** – May change is shape, position and direction as the site of origin changes and precede each QRS complex
  PR Interval - May vary along with the changes in the P wave.
- **QRS Complex –** Normal

Hemodynamic effect
None

Treatment

None required if patient asymptomatic. If rate slows, Atropine can be used.

Example of Wandering Pacemaker



Rate – 75 beats/minute
Rhythm – Irregular
P wave – Varies in size and shape
PR Interval – Varies
QRS Complex – 0.12 second; morphology: rS

5) Sinoatrial Arrest and Sinoatrial Block

Mechanism
The SA node fails to initiate an impulse at the expected time in the cardiac cycle.

Since no impulse is initiated, neither the atria nor the ventricles are stimulated and therefore an entire PQRST complex is missing. This is called "Sinoatrial Arrest".

In other instances the impulse is initiated normally but is blocked within the SA node and never reaches the atria or ventricles. This is called "Sinoatriatrial Block."

In both instances the entire PQRST complex is missing. This may result from excessive vagal dominance of the SA node.

Characteristic features/analysis
- **Rate** –Usually slow; may be normal
- **Rhythm** – Normal (except for missing beat(s))
- **P wave** – Normal (except for missing beat(s))
- **PR Interval** – Normal (except unable to determine during missing beat(s))
- **QRS Complex –** Normal (except entire complex is missing with missing beat(s)

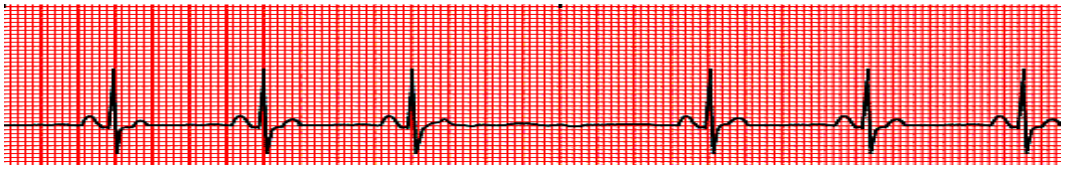
Hemodynamic effect
None if occurrence is infrequent but can produce major hemodynamic instability if frequently occurring with prolonged period of arrest or block.

Treatment
If producing signs and symptoms of low cardiac output:
Nursing intervention: Assess patient's medication profile to identify possible drugs with known bradycardia effects

Pharmacologic treatment includes: Atropine and Dopamine

Electrical therapy: application of TCP or insertion of temporary transvenous pacemaker

Example of Sinoatrial Block or Sinoatrial Arrest



Rate – 75 beats/minute, dropping to 38 beats/per minute
Rhythm – Irregular due to SA arrest (1 PQRST missing)
P wave – Regular
PR Interval – 0.16 seconds
QRS Complex – 0.06 seconds; morphology: Rs

Arrhythmias Originating in the Atria

Although the SA node is the primary pacemaker of the heart, other areas also have the ability to initiate impulses. Because the SA node normally discharges at a faster rate than the other sites, it remains the primary pacemaker.

Atrial arrhythmias result primarily from irritability of the atrial muscle, usually caused by ischemia damage or over-distention of the atrial wall. Atrial rhythm disturbances associated with a rapid ventricular rate are categorized as major arrhythmias because they increase myocardial oxygen demand and also reduce pumping efficiency of the heart.

1) Premature Atrial Contractions

Mechanism

An ectopic focus in the atrium discharges before the SA node. Because the impulse (ectopic beat) arises outside the SA node, the wave of the ectopic beat is abnormally shaped. Conduction from the AV node to the remainder of the conduction system is not affected and so the QRS complex remains normal

Note: The abnormal impulse from the atria is not a P wave. P wave is referred to the impulse originating from the SA node only

Following the premature beat there is a slight pause before the next normal sinus beat called compensatory pause. Premature atrial contractions usually reflect irritability of the atria and are a precursor of atrial fibrillation.

Characteristic features/analysis

- **Rate** –Usually normal
- **Rhythm** – Normal except ectopic beat arrives early and there is a compensating pause prior to the next normal beat.
- **P wave** – Normal except for the ectopic beat has an abnormally-shaped wave in front of the QRS complex
- **PR Interval** – May be altered on ectopic beat
- **QRS Complex –** Normal

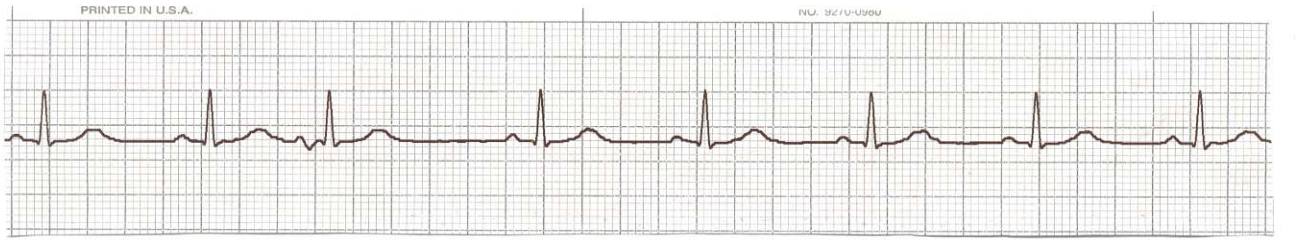
Hemodynamic effect

None if occurrence is infrequent. Frequent early diastolic PACs tend to be symptomatic than late diastolic PACs.

Treatment

Not necessary if PACs are occasional. Digoxin or Verapamil, if persistent.

Example of Premature Atrial Contractions



Rate – atrial -60/min, ventricular – 70/min
Rhythm – Irregular due to ectopic beat
P wave – Normal
PR Interval – .0.16 seconds
QRS Complex – 0.10 seconds, morhology: qRs
Interpretation – Sinus Rhythm with a Premature Atrial Contraction

2) Atrial Tachycardia (Supraventricular Tachycardia or SVT) or narrow-QRS complex tachycardia

Mechanism
Caused by the rapid discharge of an ectopic focus in the atria which occurs at a regular rate and it usually 150 – 250 beats per minute. This ectopic focus supercedes the SA node as pacemaker. The ventricles respond to each impulse and therefore the atrial and ventricular rates are identical.

Atrial tachycardia that starts abruptly and stops abruptly is termed paroxysmal atrial tachycardia or PAT

Note: Supraventricular tachycardia is caused by impulses that originate from supraventricular pacemaker (above the Bundle of His) at a rate of 150-250/min. P waves cannot be positively identified either because they merge with preceding T waves or because they are buried in the QRS complexes so the differentiation between the atrial and junctional tachycardia is impossible. QRS complexes are narrow or normal usually less than 0.11 seconds; occasionally with RBBB configuration due to aberrancy. The goal is to terminate the tachycardia immediately due to the negative hemodynamic effects to the cardiac output.

Characteristic features/analysis
- **Rate** –150-250 bpm
- **Rhythm** –.regular
- **P wave** – NA
- **PR Interval** – NA
- **QRS Complex –** Normal

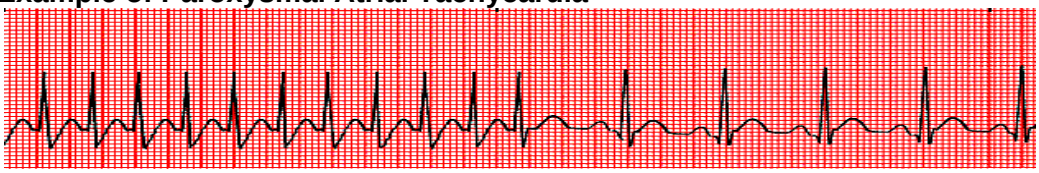
Hemodynamic effect
- May produce cardiac or chest pain due to:
  - shortened diastolic time resulting in reduced coronary perfusion time
  - shortened filling time resulting in reduced stroke volume (cardiac output)
  - increased workload resulting in increased oxygen demand

Treatment:
Nursing intervention: Initially, vagal stimulation should be attempted i.e. coughing or valsalva maneuver. (Carotid massage may be performed by the physician).

Pharmacologic treatment includes: Adenosine, Verapamil, Cardizem, Digoxin, Metoprolol

Electrical therapy: Cardioversion may be performed if arrhythmia persists

Example of Paroxysmal Atrial Tachycardia



Rate – SVT – 250 beats/min; sinus tachycardia – 115 beats/min
Rhythm – Regular
P wave – present in the sinus tachycardia but not identifiable in the SVT
PR Interval – Normal in the sinus tachycardia 0.16 seconds
QRS Complex – 0.06 seconds; morphology: Rs in SVT; qRs in ST
Interpretation – Paroxysmal Atrial Tachycardia (Supraventricular Tachycardia)

3) Atrial Flutter

Mechanism
Caused by a rapid ectopic atrial focus. The rate is usually 250 – 400 beats per minute.

The AV node is unable to conduct all of these impulses but usually allows every second, third or fourth impulse to reach the ventricles and cause a contraction.

The ventricular rate is determined by the extent of the block in the AV node.

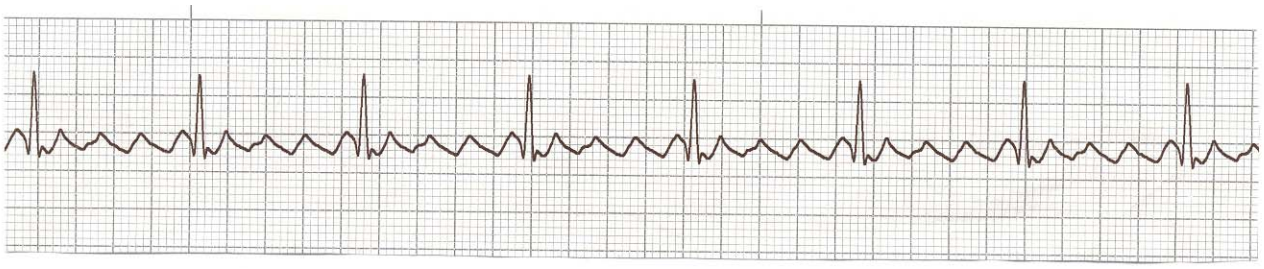
Characteristic features/analysis
- **Rate** –Atrial is 250 – 400 per minute; ventricular may range from 60 – 160/minute
- **Rhythm** – regular but may be irregular
- **P wave** – none; abnormal atrial foci described as "sawtooth" waves; called F waves or flutter waves
- **PR Interval** – NA
- **QRS Complex –**Normal

Hemodynamic effect
May produce signs and symptoms of low cardiac output due to loss of atrial contribution/contraction (atrial kick)
(The loss of atrial contraction can cause a 15 – 30% reduction in cardiac output).

Treatment
Not always necessary, however,initial treatment is usually cardioversion at lower joules.

Pharnacologic therapy: Digoxin, Verapamil

Example of Atrial Flutter



Rate – Atrial = 300/minute Ventricular = 71/ minute
Rhythm – regular
P wave – NA; Flutter waves
PR Interval –NA
QRS Complex – 0.10 seconds; morphology: qRs
Interpretation – 4:1 Atrial Flutter

4) Atrial Fibrillation

Mechanism
Ectopic foci in the atria discharge impulses at a rate of 400 – 500 per minute. The atrial muscle responds in a chaotic fashion causing the atria to fibrillate.

These extremely rapid and irregular impulses from the atria bombard the AV node. The AV node can conduct only a small percentage of these impulses to the ventricles; the remainder of the impulses are blocked.The impulses that pass through the AV node do so at irregular intervals, creating an irregular ventricular rhythm.

When the ventricular response is greater than 100 per minute, the arrhythmia is classified as "uncontrolled" atrial fibrillation, whereas a ventricular response rate of 100 beats per minute or less is classified as "controlled" atrial fibrillation.

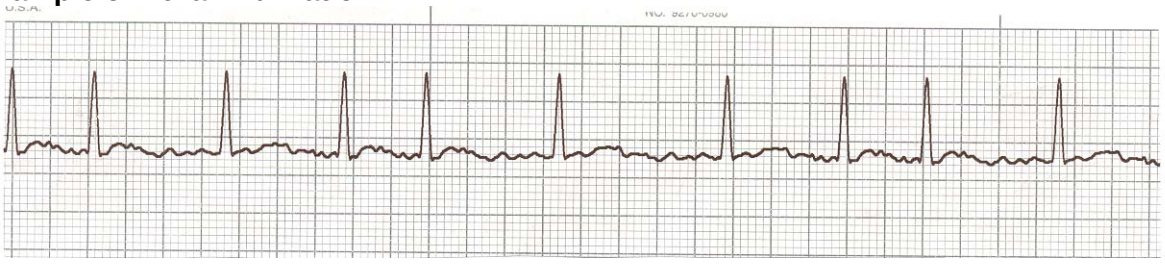
Characteristic features/analysis
- **Rate** –Ventricular rate may be normal. Rate >100 indicates fast response. Rate <100 indicates slow response.
- **Rhythm** –irregular
- **P wave** – Not present; abnormal atrial foci described as fibrillatory waves
- **PR Interval** – NA
- **QRS Complex –**Normal but occur at irregular intervals

Hemodynamic effect
Atrial fibrillation is a dangerous arrhythmia from a hemodynamic standpoint where the loss of atrial contraction can cause a 15 – 30% reduction in cardiac output and especially when the ventricular rate is rapid (uncontrolled AF causes reduction in filling time resulting in low cardiac output).

Treatment
Treatment depends on the ventricular rate, the duration of the arrhythmia and the amount of circulatory insufficiency.
Pharmacologic therapy **(directed at rate control and prevention of clot formation):** Digoxin, Verapamil, Cardizem. Aggressive anticoagulation with ASA, LMWH or Unfractionated Heparin, Clopidogrel, Warfarin. Dabigatran

Electrical Therapy: Electrical Cardioversion with anticoagulation if <48 hours. If AF is over 48 hours, anticoagulation for several weeks before cardioversion is recommended

Example of Atrial Fibrillation



Rate – Atrial = 400 or greater/ minute; Ventricular = 100/ minute
Rhythm – Irregular
P wave – None; (Fibrillatory Waves)
PR Interval – NA
QRS Complex – 0.08 seconds; morphology: R

Arrhythmias Originating in the AV Node/junction area (Nodal or Junctional rhythms)

The AV node itself does not initiate impulses. However, the junction tissue around the AV node does. Therefore, nodal arrhythmias are now termed "junctional arrhythmias". Under normal circumstances the AV junction discharges impulses only when its inherent rate is faster than that of the SA node or atria. This usually occurs because the higher centers are suppressed temporarily, allowing the normally slower AV node/junction to take over as pacemaker. When this occurs, the resulting arrhythmia is described as a junctional rhythm or junctional escape rhythm.

Like the atria and ventricles the AV junction can also produce premature beats. Most junctional arrhythmias should be treated as major arrhythmias.

1) Premature Junctional Contractions

Mechanism

A premature junctional contraction is similar to a premature atrial contraction except that the impulse originates from an ectopic focus in the AV junction area. The impulse is transmitted downward through the conduction system and produces a normal QRS complex which occurs earlier than expected in the cardiac cycle. The impulse is also transmitted **upward** through the conduction system to the atria (retrograde conduction) causing atrial stimulation. The shape of the **ectopic premature wave** depends on the origin of the impulse within the junction tissue:

Characteristic features/analysis

If	the impulse originates in the upper portion of the junction tissue	then	there is an inverted wave (looks like inverted P wave) preceding the QRS complex.
If	the impulse originates in middle portion of the junction tissue	then	no wave is present (could be buried within the QRS complex)
If	impulse originates in the lower portion of the junction tissue	then	there is an abnormal wave following the QRS complex

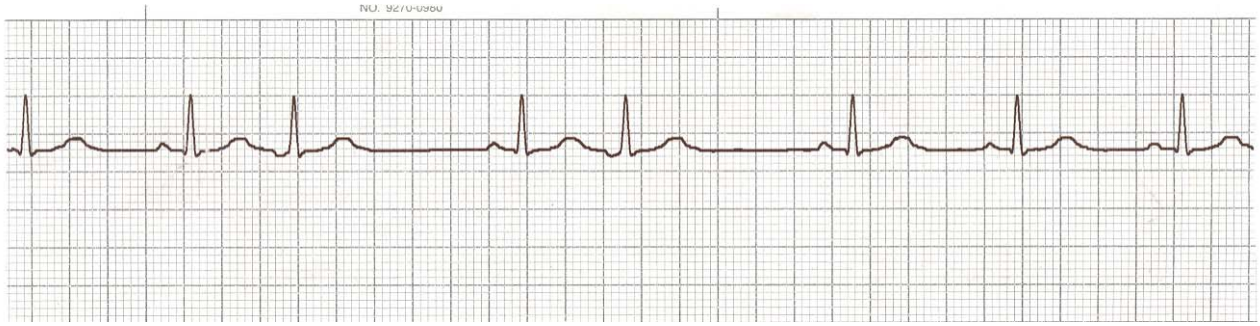
After a premature junctional beat there is an incomplete compensatory pause.

- **Rate** – Normal
- **Rhythm** – Regular except for premature beat and pause that follows.
- **P waves** –Normal and regular except for ectopic beat when abnormal wave from the AV node/junction stimulus is :
    1) inverted in front of QRS complex
    2) absent
    3) after QRS complex
- **PR Interval** – Normal for sinus beat except for ectopic beat
- **QRS Complex –** Normal

Hemodynamic effect
None if occurrence is infrequent. Frequent early diastolic PJCs tend to be symptomatic than late diastolic PJCs.

Treatment
Usually none. If patient becomes symptomatic, treat like a P.V.C.

Example of Premature Junctional Contractions



Rate – atrial – 40/min; ventricular – 60/min.
Rhythm – irregular
P wave – Normal for sinus beat
PR Interval – 0.16 second for sinus beats
QRS Complex – 0.10 seconds; morphology: qRs
Interpretation – Sinus rhythm with premature junction or nodal contractions

2) Junctional Rhythm

Mechanism
The SA node fails to initiate impulses or discharges too slowly and the AV junction area assumes control of the cardiac rhythm. *Also termed "junctional escape rhythm".

The AV junction area discharges impulses at its own intrinsic rate of 40 – 60 per minute. As with PJC's, the impulse travels down the conduction system to the ventricles and also up the conduction system to the atria. Again the abnormal stimuli waves may precede, follow, or be buried within the QRS complex. Junctional rhythms usually occur due to excessive vagal influence, ischemic damage to the SA node and drug effects (i.e. digitalis and quinidine toxicity).

Characteristic features/analysis
- **Rate** – Slow, usually 40 – 60 per minute
- **Rhythm** – Regular
- **P waves** – None. Abnormal stimuli produces waves and may:
    a) occur before the QRS complex
    b) occur after the QRS complex
    c) not be present (buried in the QRS complex)
- **PR Interval** – NA.
- **QRS Complex** – Normal

Hemodynamic effect
May produce signs and symptoms of low cardiac output due to loss of atrial contribution/contraction (atrial kick) and slow rate.
(The loss of atrial contraction can cause a 15 – 30% reduction in cardiac output).

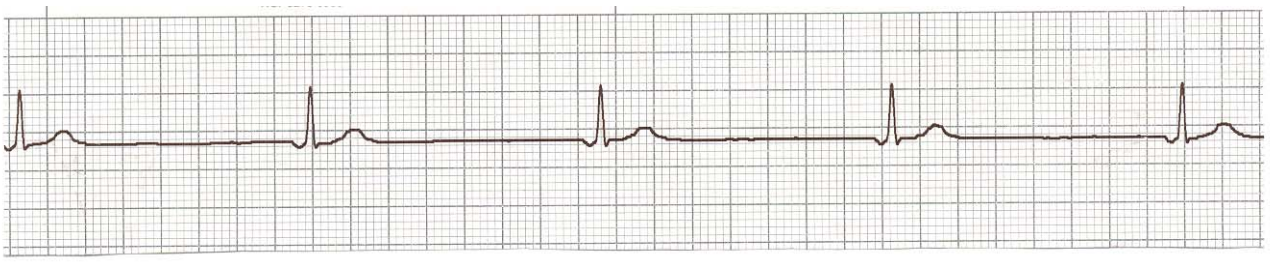
Treatment
None if patient is asymptomatic.

Nursing intervention: Observe very closely due to downward displacement of pacemaker.

Pharmacologic therapy: If it persists Atropine may be useful in allowing the SA node to resume control.

Electrical therapy: Chronotropic infusions or transvenous pacing may be necessary.

Example of Junctional Rhythm



Rate –41/minute
Rhythm – Regular
P wave – Not identified
PR Interval – Not measurable
QRS Complex – 0.08 seconds; morphology: Rs
Interpretation – Junctional rhythm

3) Accelerated Junctional Rhythm and Junctional Tachycardia

Mechanism (the mechanism for both arrhythmias are similar; the difference is in the rate) Accelerated Junctional Tachycardia exists due to an irritable focus in the AV junction area replacing the SA node as the pacemaker. Since the normal intrinsic rate of the AV Junction area is 40 – 60 per minute, the rate for an **accelerated junctional rhythm is 70 – 130 per minute**. **Junctional Tachycardia is then a rate greater than 140 per minute also called supraventricular tachycardia or narrow-QRS complex tachycardia).**

The cause of this arrhythmia is usually ischemia secondary to heart failure causing downward displacement of the pacemaker.

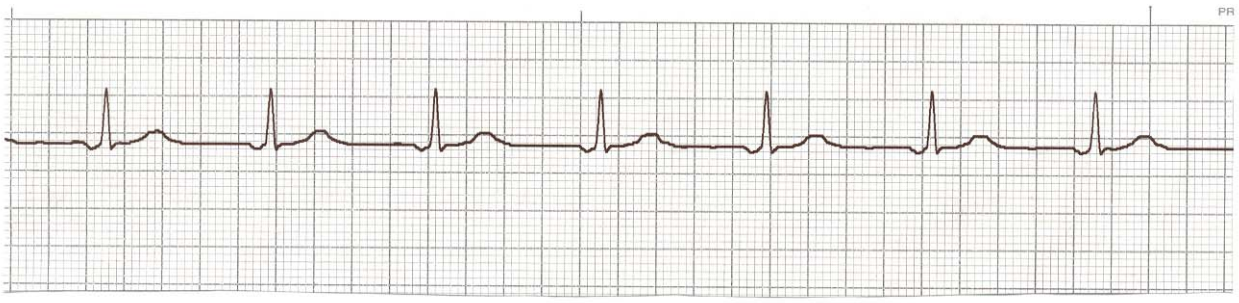
Characteristic features

- **Rate** – Accelerated junctional tachycardia - 70 – 130/minute
  - Junctional tachycardia (SVT) -140 – 250/minute
- **Rhythm** – Regular
- **P waves** – None but abnormal stimuli foci may:
  a) occur before the QRS complex
  b) occur after the QRS complex
  c) not present (buried in the QRS complex)
- **PR Interval –**NA
- **QRS Complex –** Normal

Hemodynamic effect
Usually no decrease in cardiac output is seen due to adequate heart rate. The downward displacement of the pacemaker of the heart (AV node/junction tissue as the pacemaker), makes these rhythm originating from the AV node/junction area a major rhythm

Treatment
Electrical therapy: Chronotropic infusions or transvenous Pacing.
IntraAortic Balloon Pump.

Example of Accelerated Junctional Rhythm



Rate – 71 beats/minute
Rhythm – Regular
P wave – None; abnormal stimulus inverted wave in front of QRS complex
PR Interval – NA
QRS Complex – 0.08 seconds; morhology: Rs
Interpretation – Accelerated Junctional Tachycardia

4) Junctional Tachycardia or Supraventricular tachycardia (narrow-QRS complex tachycardia

Mechanism
Junctional Tachycardia is similar to Atrial Tachycardia except that in JT the ectopic focus is in the AV node/junction rather than the atria.
If both arrythmias begin and end suddenly, the term 'paroxysmal' is added to the name of the arrhythmia i.e PJT or PAT. Impulses travel down the conduction system to the ventricles and up the conduction system to the atria. As with other junction arrhythmias, the abnormal stimuli waves may precede, follow or be buried within the QRS complex. Junctional Tachycardia may develop due to ischemia to the AV node/junction area, catecholamine release or drug induced effects.

Note: Supraventricular tachycardias are caused by impulses that originate from supraventricular pacemaker (above the Bundle of His) at a rate of 150-250/min. P waves cannot be positively identified either because they merge with preceding T waves or because they are buried in the QRS complexes so the differentiation between the atrial and junctional tachycardia is impossible. QRS complexes are narrow or normal usually less than 0.11 seconds; occasionally with RBBB configuration due to aberrancy. The goal is to terminate the tachycardia immediately due to the negative hemodynamic effects to the cardiac output.

Characteristic features/analysis
- **Rate** –Usually 140 – 250 per minute
- **Rhythm** – Regular
- **P waves** – None but abnormal stimuli waves may:
  a) occur before the QRS complex
  b) occur after the QRS complex
  c) not present (buried in the QRS complex)
- **PR Interval –**NA
- **QRS Complex –** Normal or narrow-QRS complex

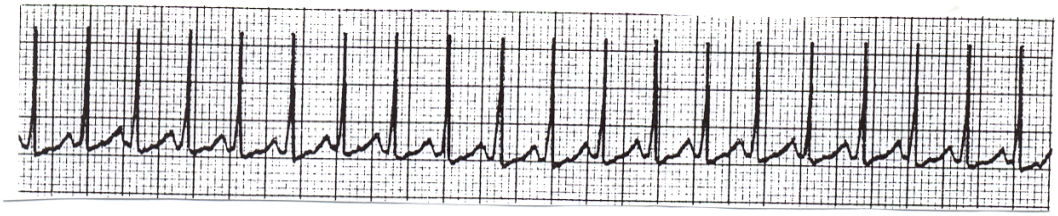
Hemodynamic effect
May produce cardiac or chest pain and other signs and symptoms of low cardiac output due to:
- shortened diastolic time resulting in reduced coronary perfusion time
- shortened filling time resulting in reduced stroke volume (cardiac output)
- increased workload resulting in increased oxygen demand
- loss of atrial kick

Treatment
The goal of treatment is to slow down the rhythm which will reveal the origin of the rhythm.
Nursing intervention: Vagal stimulation i.e. Valsalva Maneuver, coughing, carotid massage (performed by physician only).

Electrical therapy: Emergency cardioversion if sustained and symptomatic

Pharmacologic therapy: IV betablocker i.e. Metoprolol, IV Calcium Channel Blockers i.e Verapamil, IV Adenosine, IV digoxin

Example of Junctional Tachycardia



Rate – 188
Rhythm – Regular
P wave – Not identified
PR Interval – Not measurable
QRS Complex – 0.06; morphology: Rs
Interpretation – Junctional tachycardia or SVT (narrow-QRS complex tachycardia)

Arrhythmias Originating in the Ventricles (Ventricular rhythms)

Disturbances in the electrical conduction of the heart which occur in the SA node, Atria or AV node/junction area are classified as Supraventricular arrythmia. If the impulse originates in the ventricles (below the level of the AV node) the arrythmia is classified as a ventricular arrythmia.

1) Premature Ventricular Contractions

Mechanism
The most common Ventricular Arrhythmia is the Premature Ventricular Contraction (PVC). A PVC occurs when an irritable focus in the ventricles discharges an impulse before the arrival of the next anticipated impulse from the SA node or other supraventricular pacemaker. The ectopic focus stimulates the ventricles without traveling down the normal conduction system and therefore produces a wide and distorted QRS complex. The SA node is usually not affected and continues to discharge; however, the P wave is buried within the QRS complex. Following the PVC, there is a complete compensatory pause before the next stimulus comes along to initiate another impulse.

Premature Ventricular Contractions (PVCs) usually represent a sign of myocardial irritability secondary to ischemia, and the frequency and type of PVC is probably a fair index of the degree of ischemia. PVCs may initiate repetitive ventricular firing in the form of Ventricular Tachycardia (VT) or Ventricular Fibrillation (VF). Premature Ventricular Contractions are usually when they occur in one of the following forms with symptoms (patient showing signs and symptoms of low cardiac output):

- Frequently: More than 6 per minute
- Bigeminy (every second beat is a PVC)
- R on T pattern (when the PVC falls on the T wave)
- Multifocal PVCs (when the PVCs originate from more than one focus)
- Couplets or pairs (2 consecutive PVCs)

PVC is a precursor of ventricular tachycardia (VT)

Characteristic features/analysis

- **Rate** – Usually normal, but PVCs can occur at any rate.
- **Rhythm** – Regular except that when the PVC occurs, the compensatory pause creates an irregularity.
- **P waves** – Absent with the PVC
- **PR Interval** – No measurable interval with a PVC
- **QRS Complex –** The QRS Complex is always widened and distorted in shape. The exact configuration or morphology of the QRS complex depends on the site of the ventricular focus (PVC originating from the LV or PVC originating from the RV) which can be determined in lead V_1 or V_5 or V_6

Hemodynamic effect
. PVCs occurring during early diastole (non-perfused) may cause patient to become symptomatic. If PVCs occurr during late diastole (perfused), the patient will most likely be asymptomatic.

Treatment

Treatment is directed to the cause(s) of PVC.

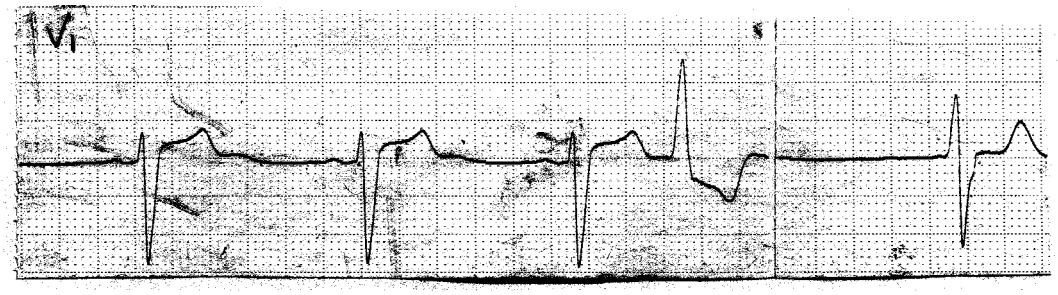
Nursing intervention: Assessment of possible causes of PVCs i.e. hypokalemia, acidosis, anemia, bradycardia, hypoxemia

NOTE: PVCs often occur in the presence of hypokalemia. Serum potassium levels should be checked when this arrhythmia occurs

Pharmacologic therapy: Primary drug therapy is Amiodarone (cordarone, IV & oral)). Other antiarrhythmics include IV Xylocaine (Lidocaine) and Procainamide (Pronestyl, IV and oral), Rythmodan (oral)

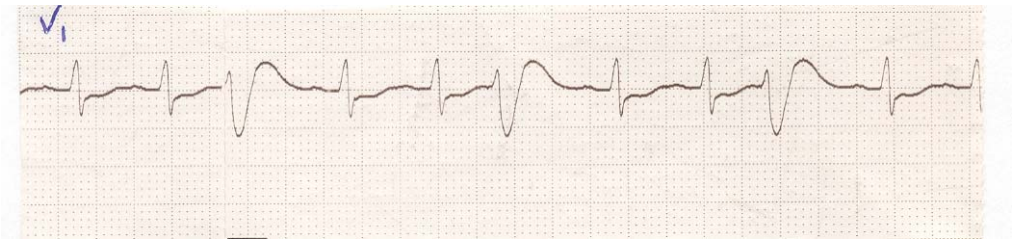
Examples of Premature Ventricular Contractions

- **A**



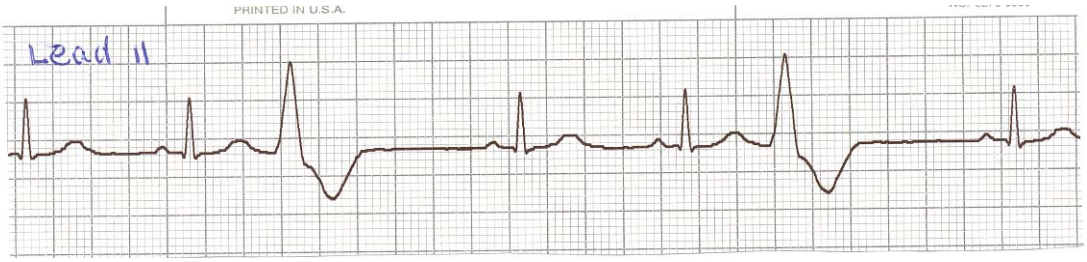
Interpretation – Sinus rhythm with left ventricular PVC

- **B**



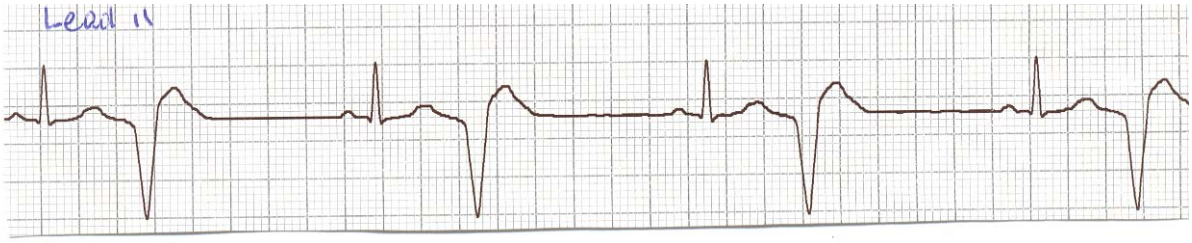
Interpretation – Sinus tachycardia with right ventricular PVCs

- **C**



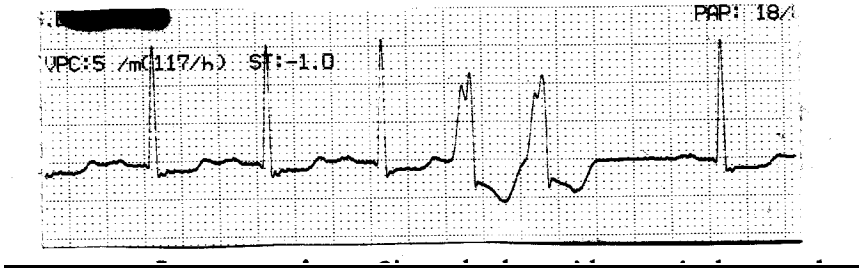
Interpretation – Sinus rhythm with unifocal PVCs

- **D**



Interpretation – Sinus bradycardia with PVCs in bigeminy

- **E**



Interpretation – Sinus rhythm with ventricular couplets

2) Ventricular Tachycardia (wide-QRS complex tachycardia)

Mechanism
Ventricular Tachycardia (VT) is described as a series of 3 or more PVCs occurring at a rapid rate. This is due to a repetitive discharge of impulses from an irritable focus within the ventricle. The irritable focus becomes the dominant pacemaker of the heart and controls the rhythm but the SA node continues to discharge at the same time. There is a dissociation of P waves and the ventricles. Ventricular Tachycardia may develop without any warning and may terminate spontaneously.

Characteristic features/analysis

- **Monomorphic VT**
  - **Rate** – Usually 140 – 220 per minute, may be even faster
  - **Rhythm** – Usually regular with even baseline
  - **P waves** – Cannot be identified, if present, are dissociated from QRS complexes
  - **PR Interval** – NA
  - **QRS Complex** – Widened and slurred

- Polymorphic VT (Torsade des Pointes)
  - **Rate** – Usually 140 – 220 per minute, may be even faster
  - **Rhythm** – irregular with undulating baseline
  - **P waves** – NA
  - **PR Interval** – NA
  - **QRS Complex** – Widened and slurred

Hemodynamic effect
Monomorphic VT: Patient may be conscious signs and symptoms of low cardiac output or unconscious (sudden cardiac arrest) due to:

- shortened diastolic time resulting in reduced coronary perfusion time
- shortened filling time resulting in reduced stroke volume (cardiac output)
- increased workload (increased heart rate) resulting in increased oxygen demand

Polymorphic VT**:** patient is unconscious with no vital signs (sudden cardiac arrest)

Treatment
Treatment is dependent on the patient's tolerance of the arrhythmia. Ventricular Tachycardia may cause severe compromise of cardiac output and cause decreased level of consciousness even to the point of unconsciousness.

Specific treatment should be as follows:

a. If the patient is conscious, has a palpable pulse and normal blood pressure, proceed as follows:

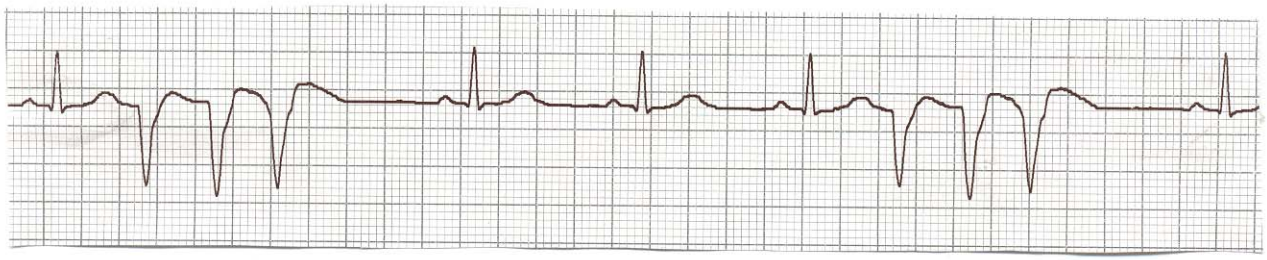
1) Ask the patient to cough several times or vagal stimulation i.e. Valsalva maneuver.
2) Give Amiodarone according to Life-saving drug (LSD) medical directive (see LSD certification program)
3) Inform the physician.
4) Continue to monitor vital signs and patient's condition.

b. If the patient is unconscious:

1) Call for help, assess for pulse and respirations
2) Start effective chest compressions if no pulse present and follow the hospital Code Blue policy and procedure
3) AED or perform manual defibrillation.
4) Chest compression should be resumed and continued between attempts to defibrillate
5) Use of LSDs during chest compressions i.e. Epinephrine and Amiodarone according to Medical Directive (see LSD certification program)
6) Inform the physician.

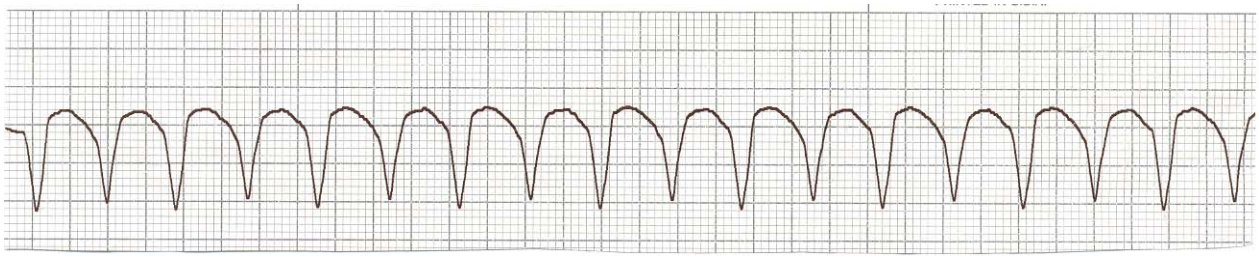
Example of Monomorphic Ventricular Tachycardia

- **A.**



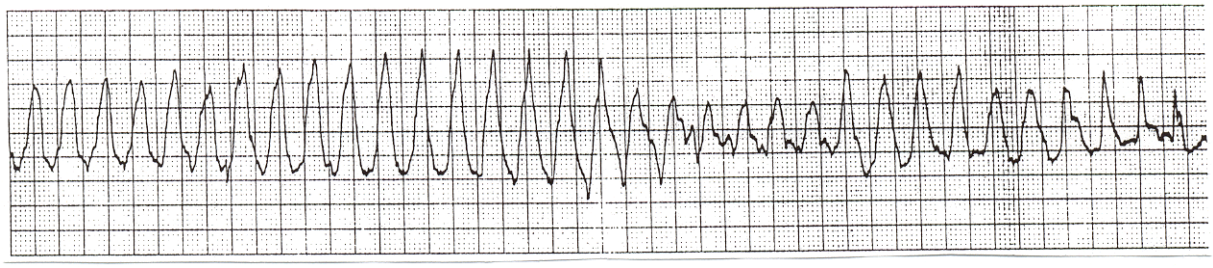
Interpretation – Sinus rhythm with paroxysmal or intermittent VT (or commonly described as short runs of VT)

- **B.**



Interpretation – (sustained) Ventricular Tachycardia

Example of Polymorphic Ventricular Tachycardia



Interpretation – Torsade de Pointes

3) Accelerated Idioventricular Rhythm

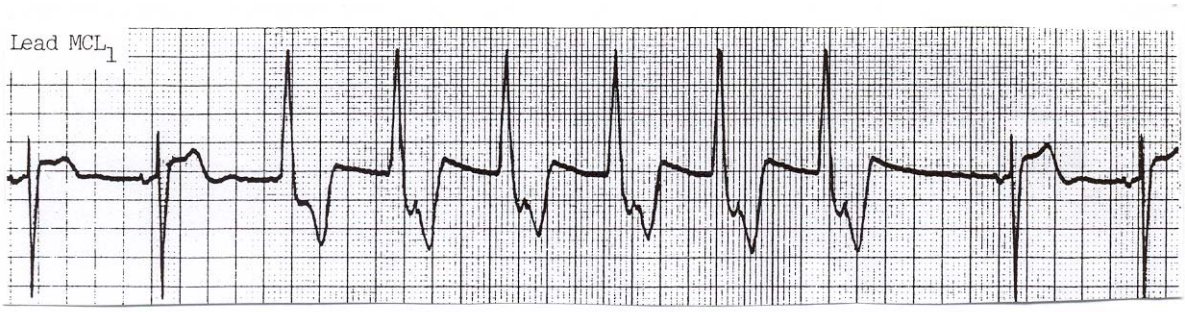
Mechanism
Accelerated idioventricular rhythm (AIVR) is similar to VT in that it is characterized by a series of consecutive ventricular ectopic beats. The difference is that the rate is only 40 – 100 beats per minute. In the case of accelerated idioventricular rhythm, the ventricles initiate their own impulse without stimulation from a higher pacemaker. The ventricles initiate impulses at a rate faster than their own instrinsic rate but not fast enough to be classified as VT. The ventricular pacemaker can only take command if its rate exceeds that of the SA node or supraventricular pacemaker. The most common cause of AIVR is ischemia to the SA node.

Characteristic features/analysis
- **Rate** – 40 – 100/minute
- **Rhythm** – Regular
- **P waves** – Not visible
- **PR Interval** – NA
- **QRS Complex** – Widened and distorted as with ventricular ectopic beats.

Hemodynamic effect
None usually noted, patient is aymptomatic.

Treatment
There is usually no treatment is required because the arrhythmia lasts only a brief period. If the arrhythmia does persist it can be treated with Atropine to block vagal influence of the SA node and increase its rate in order to allow it to become dominant again.

Examples of Accelerated Idioventricular Rhythm



Rate – 100/minute
Rhythm – Regular
P wave – Not present (except in Sinus beats where they are normal)
PR Interval – 0.12 seconds in sinus beats
QRS Complex – 0.12 seconds (during idioventricular rhythm)
0.06 – 0.08 (during sinus beat); morphology: rS (sinus) RS (AIVR)
Interpretation – Sinus rhythm with short run of Accelerated Idioventricular Rhythm

4) Ventricular Fibrillation

Mechanism
Ventricular fibrillation (VF) is the most common cause of sudden death. This arrythmia is triggered in most instances by PVC or VT. However, ventricular fibrillation can arise spontaneously. The individual muscle fibers which comprise the ventricular walls are normally stimulated simultaneously and contract in unison. In ventricular fibrillation, the electrical activity within each muscle cell becomes chaotic and thus the muscle cells lose their ability to contract. The impulses are discharged so rapidly that the ventricles merely twitch continuously. Since cardiac output ceases, circulation stops abruptly and death follows within minutes.

NOTE: It is safe to assume that a conscious patient does not have ventricular fibrillation.

Characteristic features/Analysis
The ECG pattern is characterized by a rapid repetitive series of "chaotic" waves originating in the ventricles. The waves have no uniformity and are bizarre in configuration. PQRST cannot be identified. The complexes differ from each other and occur in completely irregular fashion.

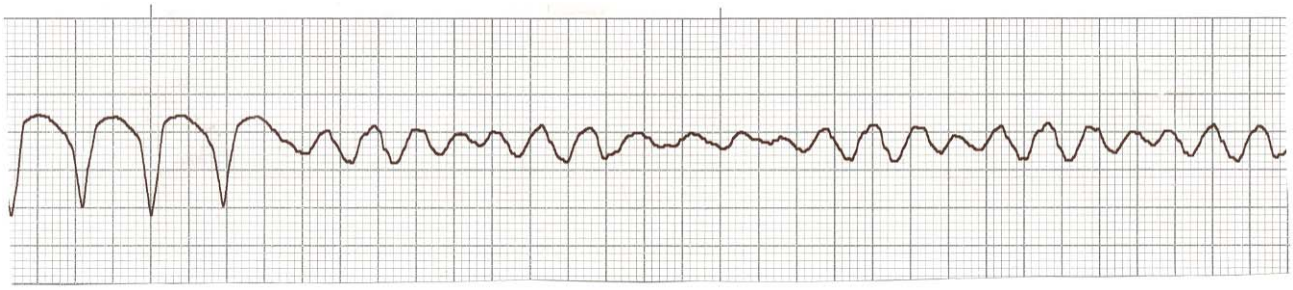
Hemodynamic effect
The chaotic waves results in cessation of blood circulation therefore absence of vital signs**.**

Treatment
Always ensure that the arrhythmia is indeed ventricular fibrillation as opposed to artifact. Because the arrhythmia is characterized by a "series of repetitive chaotic waves", it is easily mistaken for artifact. Time should not be wasted checking for possible means of artifact; instead, the patient should be assessed for level of consciousness and the presence of any palpable pulses. Once the arrhythmia is identified as ventricular fibrillation then the next step is "Termination of the arrhythmia by means of Electrical Defibrillation".

NOTE: Once it is determined that the patient is indeed demonstrating ventricular fibrillation, other life-saving measures such as C.P.R. stating with chest compressions (2005 ECC Guidelines) should be initiated until electrical defibrillation (AED or manual) is possible.

Once electrical defibrillation has successfully been carried out, chest compressions are resumed and ACLS medications such as Epinephrine and Amiodarone must be administered during chest compressions according to Medical Directive (see LSD certification program)

Example of Ventricular Fibrillation



5) **Ventricular Standstill**

Ventricular contraction depends on an effective electrical stimulus. If for some reason electrical stimuli to the ventricles are of inadequate intensity or if they cease entirely, the ventricles stop contracting with the result being ventricular standstill or ventricular asystole. The effect of ventricular standstill is the same as with ventricular fibrillation…sudden death.

Ventricular standstill may develop as a primary electrical disorder called **Primary Ventricular Standstill** or as a terminal arrhythmia called **Secondary Ventricular Standstill**.

Mechanism

In Primary Ventricular Standstill, sinus (or Atrial) impulses are discharged normally and produce P waves but all of these impulses are suddenly blocked and never reach the ventricles. The inherent ventricular pacemaker does not begin to initiate impulses and therefore there is no ventricular activity. Primary ventricular standstill is usually due to a conduction disorder.

Characteristic features/Analysis

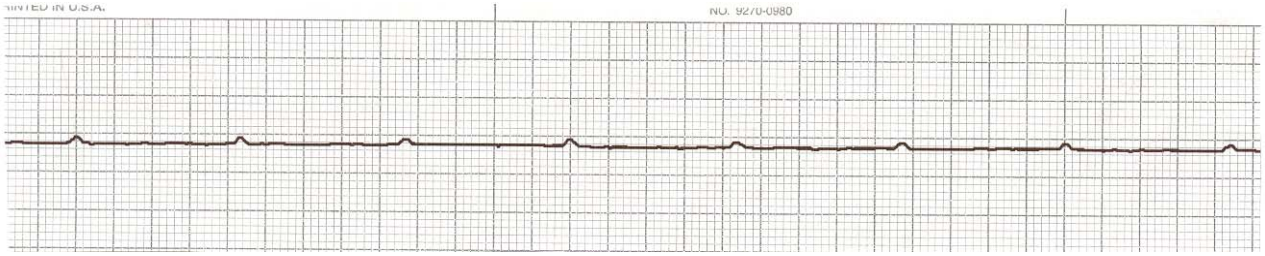
- **Rate** – P waves – rate may be normal, no palpable pulse
- **Rhythm** – regular P waves
- **P waves** – Usually normal
- **PR Interval** – No PR interval since there is no QRS complex
- QRS Complex – None

Hemlodynamic effect
Although the SA node is working, there is no ventricular contraction or activity and therefore no circulation or vital signs.

Treatment
Aimed at stimulating the ventricles to contract by:

- Initiation of CPR, starting with chest compressions (2010 ECC Guidelines)
- Epinephrine
- Cardiac Pacing

Example of Primary Ventricular Standstill



Rate – Atrial: 75 Ventricular: 0
Rhythm – Regular atrial rhythm
P wave – P waves present
PR Interval – NA
QRS Complex – Absent
Interpretation – Primary Ventricular Standstill

Secondary Ventricular Standstill (idioventricular rhythm or agonal or dying heart rhythm)

Mechanism
Secondary Ventricular Standstill is always associated with heart failure and is a terminal event. This arrhythmia is usually due to inadequate tissue perfusion resulting from hypoxia. The heart's electrical activity becomes insufficient to stimulate the myocardium; thus, ventricular standstill occurs.

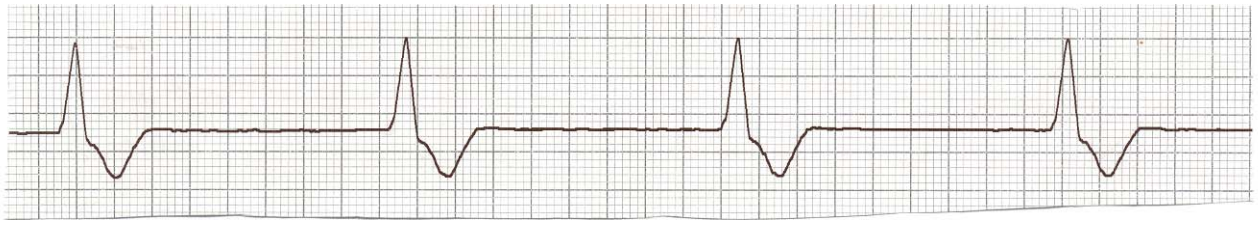
Characteristic features/analysis
- **Rate** – May have infrequent QRS complexes 15-40/minute, which indicates electrical activity still occurs but there is no mechanical contraction.
- **Rhythm** – may or may not be regular.
- **P waves** – Absent
- **PR Interval** – NA
- **QRS Complex –** Widened and slurred

Hemodynamic effect
The QRS complex is ineffective in producing mechanical contraction, thus no circulation and vital signs absent.

Treatment
This secondary type of standstill seldom yields to resuscitative measures because the oxygen-deprived myocardium is unable to respond to any stimulation. Electrical activity may continue but the myocardium does not respond.

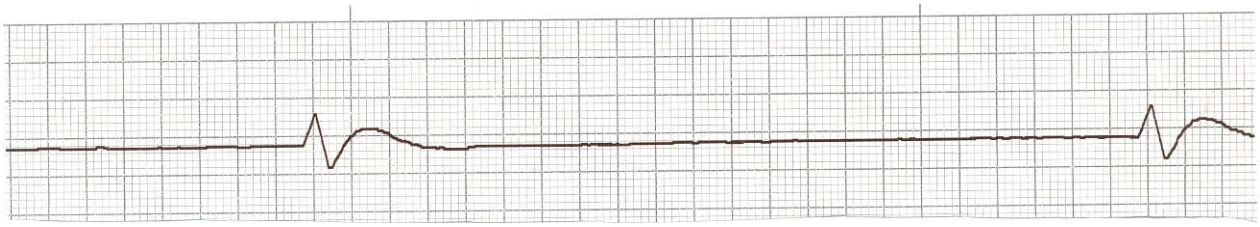
Example of Secondary Ventricular Standstill

- **A**



Rate – 35/minute
Rhythm – Regular
P wave – Absent
PR Interval – NA
QRS Complex – 0.16 seconds, morphology: Rs
Interpretation – Secondary Ventricular Standstill (Idioventricular Rhythm)

- **B**



Interpretation – Agonal rhythm

Disorders of Conduction (Heart Blocks)

Any interference or abnormal delay in the passage of impulses from the SA node through the conduction system is described as a "heart block".
Heart blocks can occur at any level of the conduction system and so may be categorized as to their main anatomical sites of involvement.

a) Blocks in the SA node or Atria
b) Blocks in the AV node or Junction area
c) Blocks in the HIS-Purkinje system (Intraventricular branches)

1. First-degree AV Heart Block

Mechanism
First-degree AV block denotes that conduction from the SA node to the ventricles is abnormally delayed within the AV node or junction area. This delay is usually due to ischemia to the AV node.

Characteristic features/Analysis

- **Rate** – Usually normal
- **Rhythm** – Regular.
- **P waves** – Normal
- **PR Interval** – Prolonged beyond 0.20 seconds
- **QRS Complex –** Normal

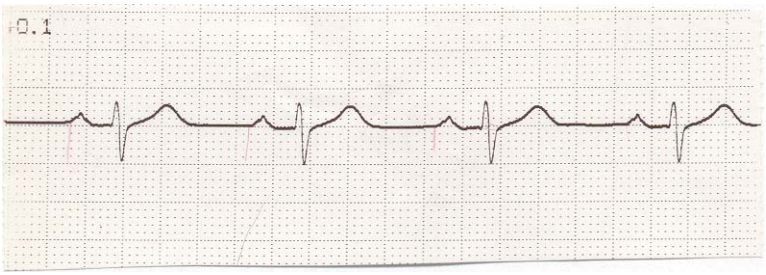
Hemodynamic effect
None. If heart rate slow, may result in low cardiac output

Treatment
Not a serious arrhythmia unless prolongation greater than 0.28 seconds.
Nursing Intervention**:** Observe for progressive lengthening of PR interval
Assess medications. Observe for 2 or 3 AV block.

Pharmacologic therapy: Atropine or chronotropic infusions for symptomatic slow rate.

Electrical therapy: Temporary pacing (transcutaneous or transvenous) may be required if unresponsive to Atropine

Example of First-degree AV Heart Block



Rate – 65/ minute
Rhythm – Regular
P wave – Normal
PR Interval – 0.24 seconds
QRS Complex – 0.12 seconds; morphology: rs
Interpretation – Sinus rhythm with First-degree AV Block

2. **Second-degree AV Heart Block Type I (Wenckebach)**

Mechanism
In second-degree AV block, some impulses from the SA node do not reach the ventricles because they are blocked within the AV node. When this occurs, a P wave is not followed by a QRS complex and a ventricular beat is missing.

Characteristic features/analysis
The most common cause of Second-degree AV Block is ischemia to the AV node.

- **Rate** – Usually slow but may be normal
- **Rhythm** – Irregular due to dropped beats
- **P waves** – The number of P waves are always greater than the number of QRS complexes due to the dropped beats.
- **PR Interval** – There is progressive prolongation of the PR interval until a sinus impulse is blocked and a QRS complex fails to appear. Following the dropped beat, the PR interval is shortened and the entire sequence starts again.
- **QRS Complex –** Normal

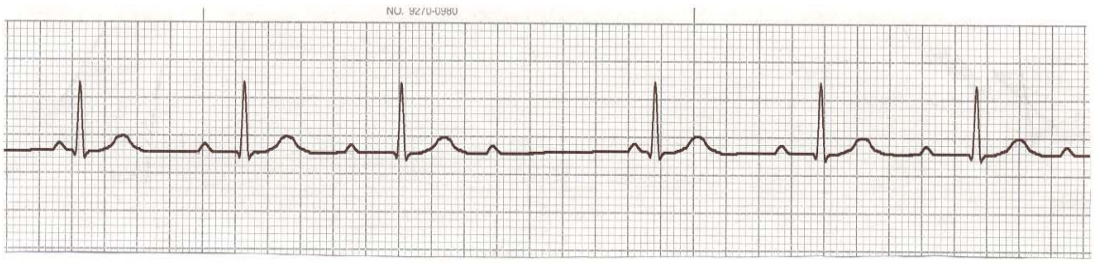
Hemodynamic effect
Usually none if heart rate is normal. Slow heart rate will sometimes result in signs and symptoms of low cardiac output.

Treatment
Depends on ventricular response rate
. Nursing intervention: Observe for deterioration of AV block.
Assess for signs and symptoms of low cardiac output.
Assess medications

Pharmacologic therapy: for symptomatic slow rate, Atropine or chronotropic infusions

Electrical therapy: Temporary pacing (transcutaneous or transvenous) if unresponsive to Atropine

Example of Second-degree AV Block Type 1 (Wenckebach)



Rate – Atrial: 70/minute; Ventricular: 60/minute
Rhythm – Irregular
P wave – Present and normal
PR Interval – 0.16 seconds progressing to 0.24 seconds, then 0.30 seconds
QRS Complex – 0.08 seconds, morphology: qRs
Interpretation – Second-degree AV block Type I (Wenckebach)

3. Second-degree AV Heart Block Type II or Mobitz

Mechanism
Second-degree AV block Type II or Mobitz is like Type I block or Wenckebach, characterized by failure of some sinus impulses to conduct to the ventricles. However, the PR intervals in Type II do not lengthen progressively before the dropped beats, instead the PR interval of the conducted beat is of constant duration (normal) and suddenly a sinus P wave is blocked.

Impulses may be blocked occasionally or at regular intervals. When the block occurs at a regular interval, like every 2nd, 3rd or 4th beat, the arrhythmia is termed a 2:1, 3:1 or 4:1 block.

Mobitz Type II block is much less common than Type I (Wenckebach), but is far more serious. This arrhythmia usually results from injury to the AV nodal area or the HIS Purkinje system.

Characteristic features/analysis
- **Rate** – Ventricular rate usually slow
- **Rhythm** – Depending on the degree of block, the rhythm may be regular, i.e. 2:1 block. If blocked beats occur occasionally the rhythm is irregular
- **P waves** – Normal in configuration but more numerous than the QRS complexes.
- **PR Interval** – Normal beats have a normal PR interval
- **QRS Complex** – Usually widened, this indicates the block is low in the AV junction.

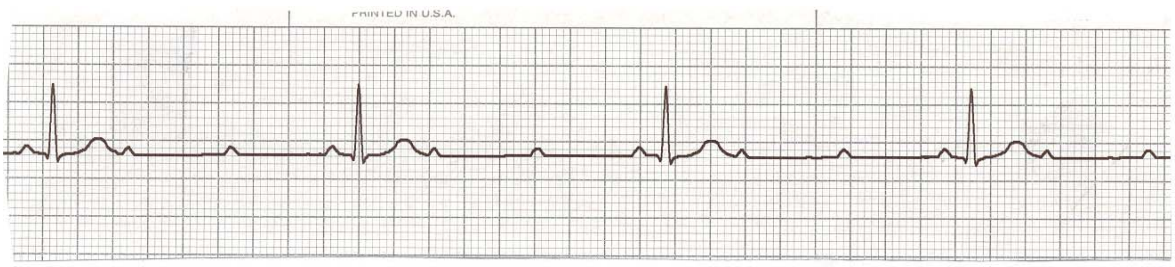
Hemodynamic effect
Patient may become symptomatic if heart rate is slow. This is a higher degree of block and therefore can result in cardiac and circulatory compromise.

Treatment
Nursing intervention: Close observation of patient. Assess medications.

Pharmacologic therapy: Dopamine infusion if hypotensive due to slow heart rate. Dopamine or Epinephrine will increase heart rate (chronotropic effect)
NOTE: Atropine is contraindicated.

Electrical therapy: Temporary pacing (transcutaneous or transvenous)

Example of Second-degree AV Heart Block Type II (Mobitz)



Rate – Atrial: 76 per minute Ventricular: 38 per minute
Rhythm – Regular
P wave – Two P waves for each QRS complex
PR Interval – 0.24 seconds when preceding a QRS complex
QRS Complex – 0.12 seconds

4. Third-degree (Complete) AV Block

Mechanism
In third-degree AV block, all impulses from the atria are blocked and none reach the ventricles; thus, the atria and the ventricles beat independently, each controlled by a separate pacemaker. The SA node serves as the pacemaker for the atrial, while the ventricles receive their impulses from a focus within the ventricles, which usually initiates impulses at a rate of 30 – 40 per minute. **There is no relationship between P waves and QRS complexes.** This condition is also termed "Atrio-ventricular Dissociation".

Characteristic features/analysis

- **Rate** – The ventricular rate is usually slow. The atrial rate is always faster than the ventricular
- **Rhythm** – Both atrial and ventricular rhythms are regular but independent of each other
- **P waves** – Normal and regular but do not reach the ventricles. There are more P waves than QRS complexes
- **PR Interval** – No relationship between P waves and QRS complexes; therefore PR interval is not significant.
- **QRS Complex –** Usually widened and distorted; depends on the origin of the ventricular Pacemaker**.**

Hemodynamic effect
Can be asymptomatic especially if good ventricular rate; atrial contribution from P waves is negligible due to asynchronous activities between P waves and QRS complexes.

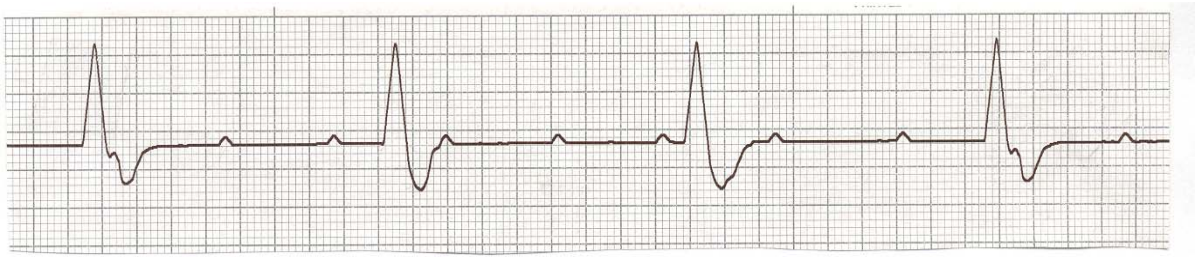
Treatment

Nursing intervention: Close observation and assessment of cardiac output. Assess medication.

Pharmacologic therapy: Dopamine infusion if hypotensive due to slow heart rate. Dopamine and Epinephrine will increase heart rate (chronotropic effect)
NOTE: Atropine is contraindicated.

Electrical therapy: Temporary pacing (transcutaneous or transvenous)

Example of Third-degree (Complete) AV Block



Rate – Atrial: 90/minute; Ventricular: 35 per minute
Rhythm – Regular
P wave – Normal, but not related to QRS complex
PR Interval – NA as no relationship between P waves and QRS complexes.
QRS Complex – 0.16 seconds, morphology: RS

Intraventricular Blocks (Bundle Branch Blocks) or Intraventricular Conduction Delays (IVCD)

Mechanism

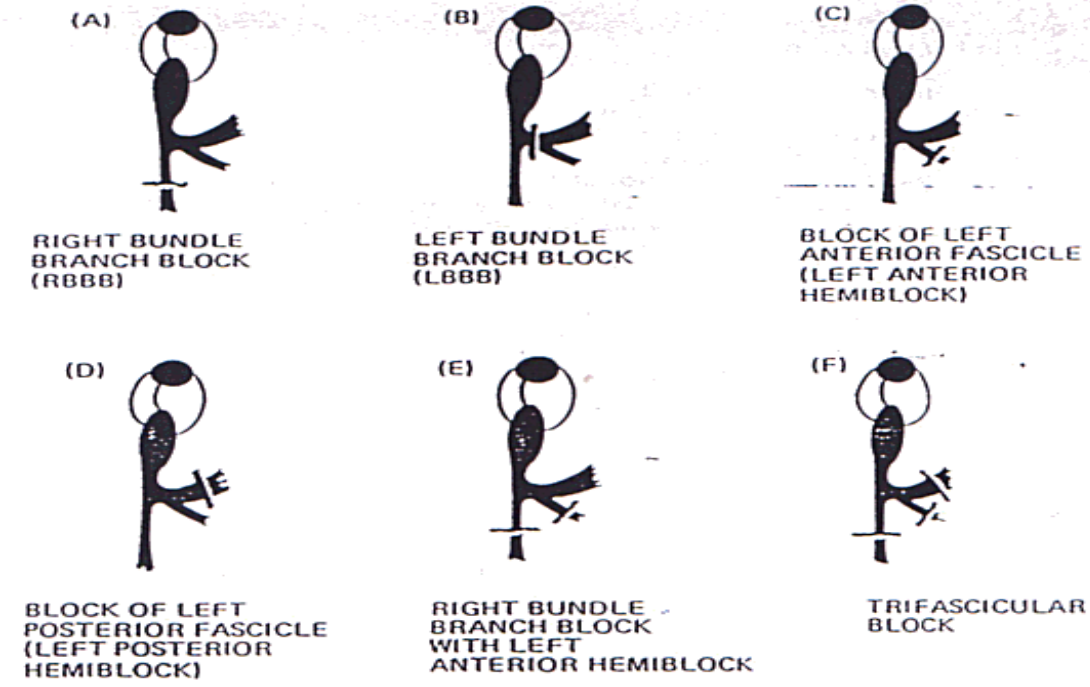
All blocks occurring below the bifurcation of the bundle of HIS are categorized as intraventricular blocks.

Sinus impulses, after traversing the AV node and bundle of HIS, are conducted down the left and right bundle branches and stimulate the respective ventricles simultaneously. The total time for depolarization of both ventricles, as represented by the width of the QRS complex is less than 0.12 seconds (.04 -.12 seconds).

When one of the bundle branches is blocked impulses travel through the intact branch and stimulate the ventricle it supplies on schedule. The impulse must travel across the intraventricular system to stimulate the ventricle affected by the blocked bundle branch. The delay caused by this change in conduction pattern is manifested on the ECG by widening of the QRS complex beyond 0.12 seconds. Bundle branch blocks usually reflect myocardial damage.

The bundle of HIS consists of 2 branches; the left bundle branch and the right bundle branch. The left bundle branch is comprised of two parts, an anterior and a posterior fascicle. Any of these branches can block individually or in combination.
Combination of blocks may be as follows

1) Block of the Right Bundle Branch (RBBB)
2) Block of the “main” Left Bundle Branch (LBBB / also bifascicular block)
3) Block of the “anterior” fascicle of the Left Bundle Branch (Left Anterior Hemiblock)
4) Block of the “posterior” fascicle of the Left Bundle Branch (Left Posterior Hemiblock)
5) Blocks involving the right bundle branch and one of the fascicles of the left bundle (Bifascicular Block)
6) Blocks involving all three branches of the bundle of HIS (Trifascicular Block)



Characteristic features/analysis (Lead V_1 (refer to advantages of Lead V1 continuous monitoring) will differentiate RBBB and LBBB

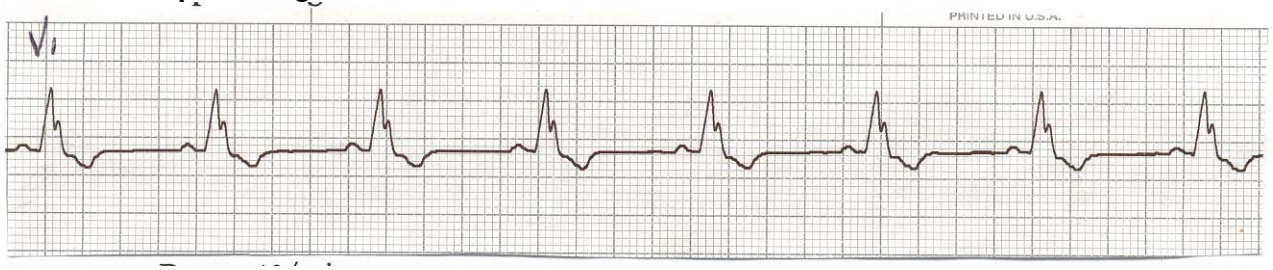
- **Rate** – Usually normal. Bundle blocks may be rate dependent.
- **Rhythm** – Regular
- **P waves** – Normal
- **PR Interval** – Normal
- QRS Complex **–** Always widened >0.12 seconds and the configuration of the complex is distorted. (After the uninvolved ventricle is stimulated the impulse must then be transmitted through the interventricular septum to activate the blocked side. This delay in activation causes the QRS complexes to be wide and notched).

Hemodynamic effect
None. Generally, the widening of the QRS complex has no hemodynamic effect**.**

Treatment
Bundle branch blocks (BBB) generally require no treatment.
Nursing intervention: Observe closely especially new LBBB in AMI. Assess medications; (overdose of antiarrhythmics may cause intraventricular blocks).

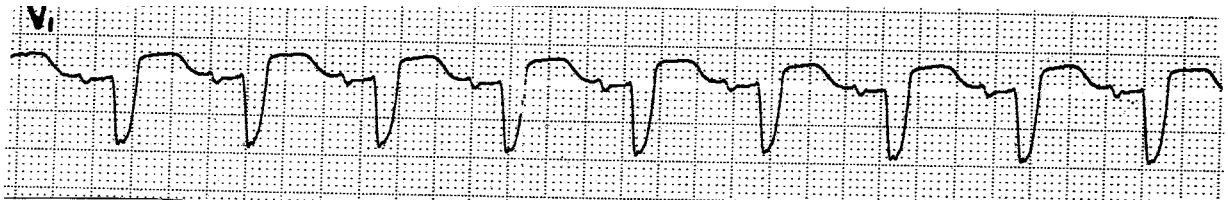
Electrical therapy: BBBs that develop acutely may require cardiac pacing.

Example of Right Bundle Branch Block



Rate – 68/minute
Rhythm – Regular
P wave – Normal
PR Interval – 0.14seconds
QRS Complex – 0.14 seconds; Morphology: RR^1 or RSR^1 with a depressed ST segment
Interpretation – Sinus Rhythm with Right Bundle Branch Block

Example of Left Branch Bundle Block



Rate –83/minute
Rhythm – Regular
P wave – Normal
PR Interval – 0.20 seconds
QRS Complex – >0.16 seconds, morphology: rQ or Qr with ST segment elevation
Interpretation – Sinus Rhythm with Left Bundle Branch Block

Paced rhythm

When the arrhythmia is too slow resulting in severe signs and symptoms of low cardiac output, an electronic device called pacemaker is implanted to help regulate the heart's rhythm. Pacemakers can be temporary (transcutaneous or transvenous) or permanent.

In an emergency, the temporary transcutaneous pacemaker (TCP) can be applied for immediate resuscitation of bradyarrhythmias or asystole. TCP should be a short term emergency measure only due to pain of pacing transcutaneously. Arrangement should be made for insertion of transvenous pacemaker.

Permanent pacemaker is inserted as a result of unresolved arrhythmia or diseased electrical conduction system i.e. AMI involving the conduction system.

Whether pacemaker is temporary or permanent, the principles of pacing the heart is the same except for some program options that can only be available in permanent pacemaker i.e. rate-responsive options where a pacemaker will deliver electrical impulses to the heart that suits a person's particular need or demand..

Pacemakers can be programmed to discharge electrical impulses at a 'fixed rate' but almost all work on 'demand'. 'On demand' means that if the pacemaker senses that the heart has missed a beat or is beating too slowly, it will discharge electrical impulses continually. However, if the pacemaker senses that the heart is beating on its own (intrinsic rate), it will not discharge any electrical impulse.

Pacemakers can be single-chamber or dual-chamber.

Single-chamber pacemaker has one lead to carry signals to and from one chamber of the heart- either the right atrium or, more commonly, the right ventricle.

A **dual-chamber pacemaker** has two leads, one in the right atrium and the other in the right ventricle.

Characteristic features/analysis

(Lead V_1 (refer to advantages of Lead V1 continuous monitoring) will identify and confirm the proper ventricular lead placement which is in the right ventricle)

- **Rate –** depends on the ordered or required rate setting
- **Rhythm -** regular
- **P waves** – may or may not be visible
- **PR Interval** – NA
- **QRS Complex –** Always preceded by a spike (electrical impulse from pacemaker); widened >0.12 seconds and the configuration of the complex is distorted. The pacemaker impulses originate in the right ventricle resulting in an abnormal ventricular depolarization; hence the LBBB QRS complex morphology. The repolarization of the abnormally depolarized ventricles will also result in abnormal repolarization, hence ST segment elevation.
  Morphology**:** rS or Qr

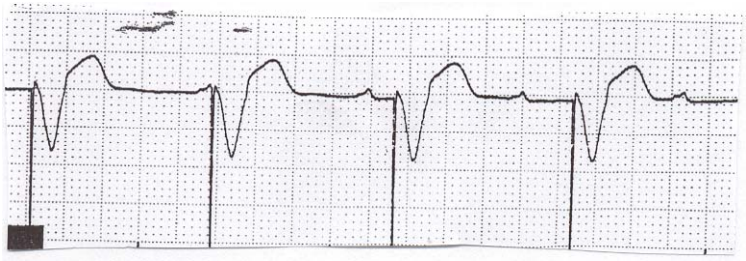
Hemodynamic effects

Loss of atrial kick from single-chamber ventricular pacemaker is compensated by increasing the pacemaker rate.

The dual-chamber pacemaker is most physiologic due to synchronization of atrial and ventricular activities (atrial kick is not lost).

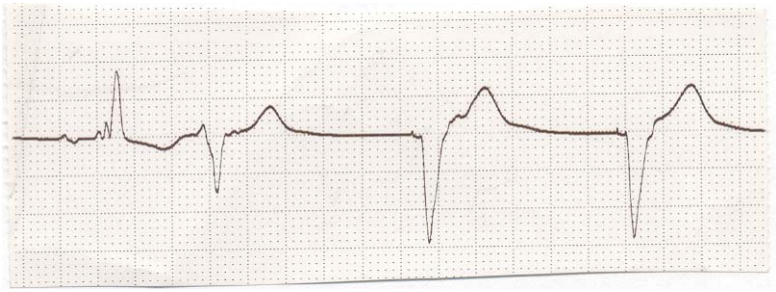
Examples of paced rhythm

- **A**



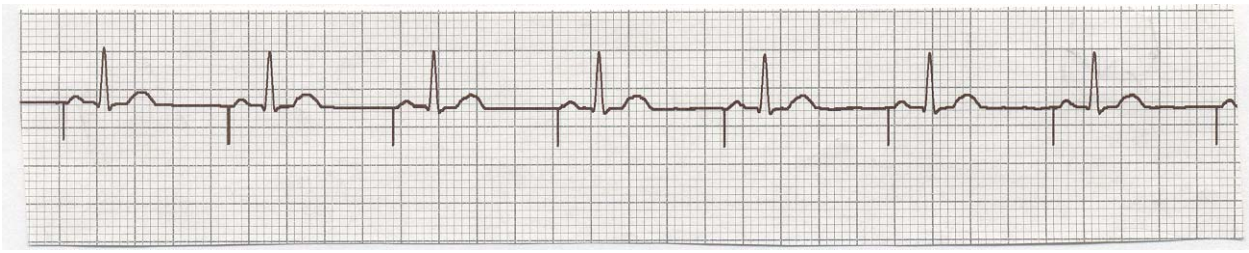
Interpretation – Ventricular paced rhythm

- **B**



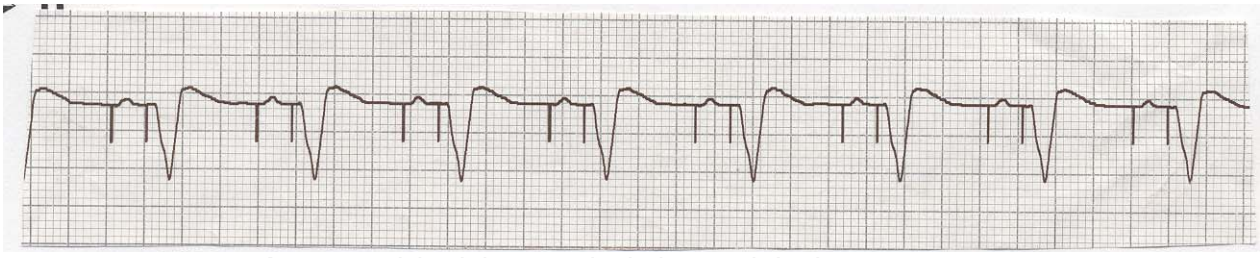
Interpretation – Ventricular paced rhythm

- **C**



Interpretation –Atrial paced rhythm

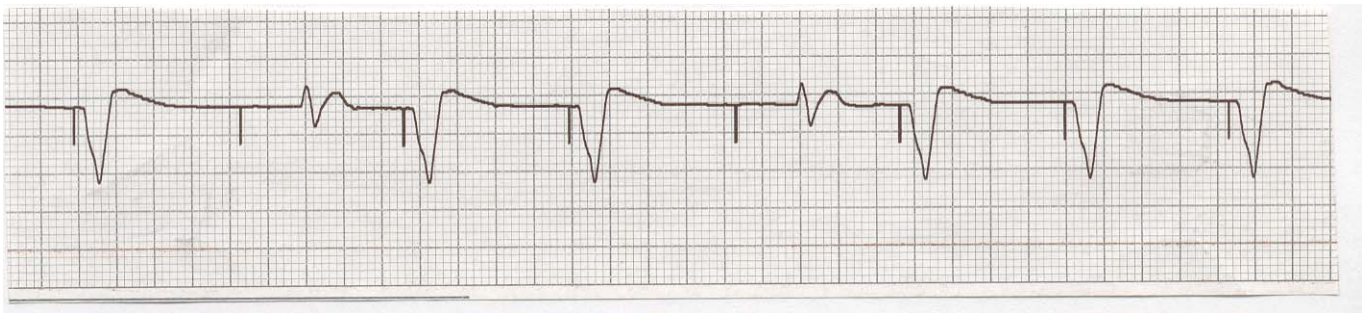
- **D**



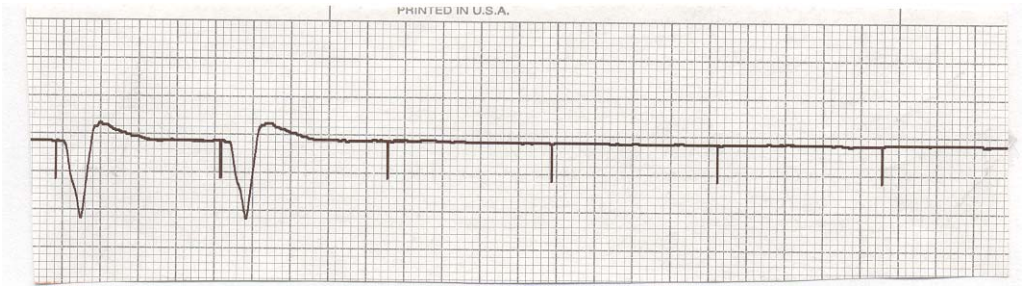
Interpretation – Dual (Atrial & ventricular) paced rhythm

What's wrong with the picture?

- **A**



- **B**



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Current Guideline References

3. 2010 ECC Guidelines, Heart and Stroke Foundation
4. Buff, J. (2012) ECG Workout: Exercises in Arrthythmia Interpretation. Philadelphia: Lippincott Williams & Wilkins

Committees/Stakeholders	Approved	Reviewed
Manager/Nurse Clinician/Educator	**12 June 2006**	
ICU Patient Care Team	14 Sept 2006	07 Nov 2013
Critical Care Program Council	22 Sept 2006	22 Nov 2013