BASIC ECG INTERPRETATION STUDY MODULE

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Purpose

The ECG module is designed to improve the ability of all levels of ECG interpreters. This module will form the foundation of your ECG knowledge and enable you to understand the components of the normal ECG.

Objectives

At the completion of this module and the cardiac rhythms workbook exercises, you will be able to:

1. Explain the cardiac conduction system
2. Apply cardiac monitoring electrodes
3. Identify cardiac rhythm waveforms
4. Interpret cardiac rhythm strips using the 6-step method
5. Interpret selected Sinus, Atrial, Junctional, and Ventricular arrhythmias
6. Interpret first, second, and third- degree Atrioventricular (AV) Blocks
7. Identify life- threatening arrhythmias and the primary nursing actions and medical treatments needed

Materials (if applicable)

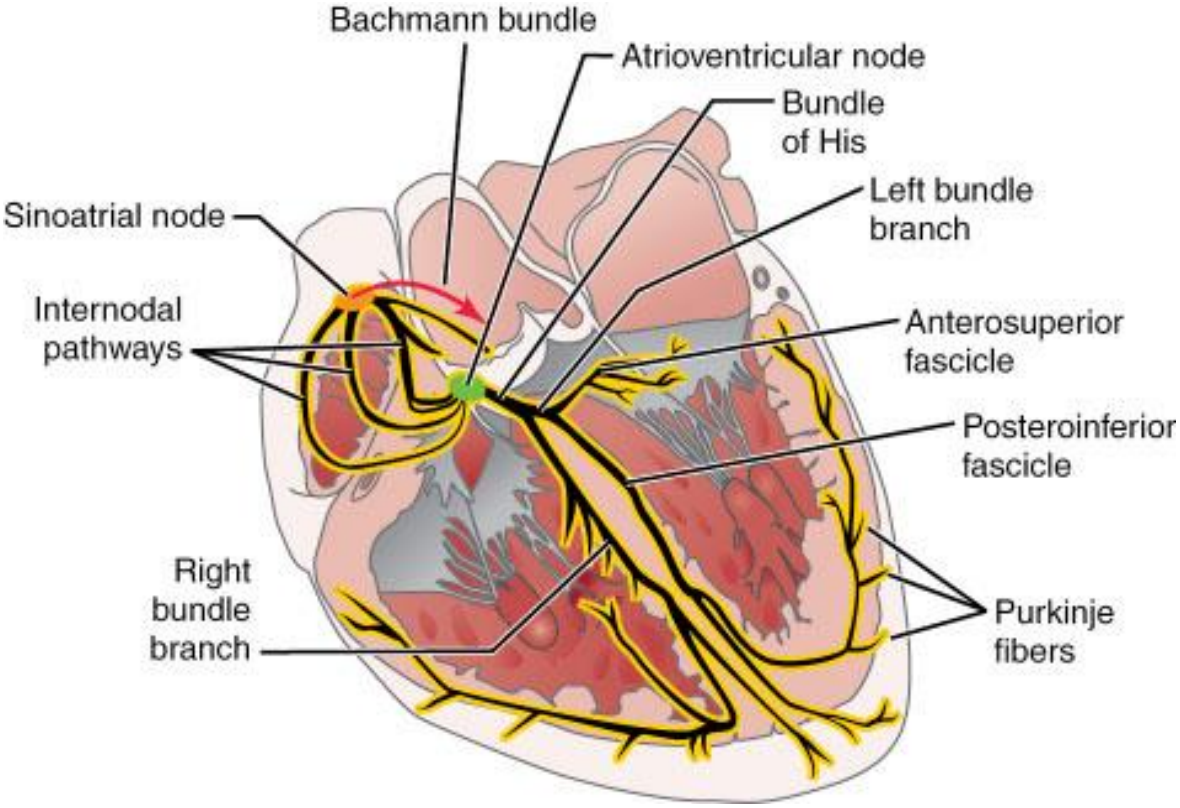
N/A

Information

ELECTROCARDIOGRAPHY

The Cardiac Conduction System

There are four main components of the cardiac conduction system, namely, the SA Node, the AV Node, the Bundle Branches and the Purkinje Fibers. In addition, subcomponents called tracts, branches or bundles complement this conduction network. Each of these components consists of specialized cardiac tissue capable of conducting electrical impulses.



Cardiac conduction system landmarks (skillstat, ‘‘n.d”)

Sinoatrial Node (SA Node) The SA node is the first or primary pacemaker of the heart and is capable of initiating 60 - 100 beats per minute (bpm). It contains specialized pacemaker cells which are located in the right atrium adjacent to the orifice of the superior vena cava.

Bachmann’s Bundle Bachmann's Bundle is the interatrial pathway which facilitates the transmission of electrical impulses from the right to the left atrium.

Internodal Tracts (Pathways) There are three internodal tracts, namely, inner, middle and outer which transmits impulses from the SA node to the AV Junction.

Atrioventricular Node (AV Node) The AV node is the second pacemaker of the heart and is capable of initiating 40 – 60 bpm. It contains specialized pacemaker cells which are located above the interventricular septum in the right atrium near the orifice of the coronary sinus ostium. The AV node regulates the number of electrical impulses which are conducted to the ventricles which permits ventricular filling during atrial contraction.

Bundle of His Thick bundle of fibers which extends down the right side of the interventricular septum and receives electrical impulses from the AV Junction then transmits them to the bundle branches.

Right Bundle Branch Descends from the Bundle of His to the right septal wall and transmits electrical impulses to the right ventricular Purkinje Fiber system.

Left Bundle Branch Descends to the left septal wall and divides into the anterior and posterior branches which transmit impulses to the left ventricular Purkinje Fiber network.

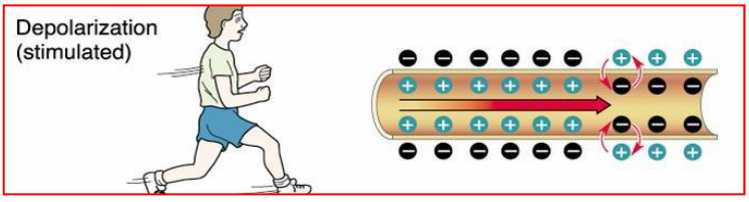
Purkinje Fibers (Purkinje Network or Purkinje System) The Purkinje Fibers are the lowest pacemakers of the heart and are capable of initiating less than 40 bpm. This network of fibers is contained throughout the inner surface of both ventricles and stimulates the myocardial cell.

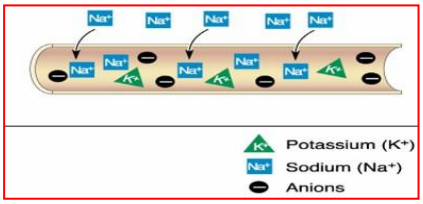
Ectopic Pacemakers Ectopic pacemakers are cells which are located outside the normal conduction pathways which are capable of initiating electrical impulses. They are essentially contractible cells located in the atria, ventricles and at the AV Node fibers.

Cardiac Electrophysiology

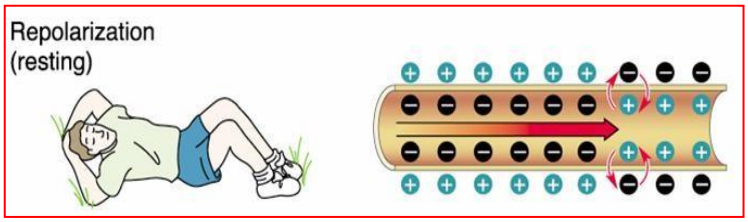
Before the heart can mechanically contract and pump blood, cardiac muscle cell depolarization must take place. A difference between electrical charges must exist in order for electrical current to be generated. The major electrolytes that affect cardiac function are Sodium (Na+), Potassium (K+), and Calcium (Ca+2). The exchange of electrolytes in myocardial cells creates electrical activity, which appears on the ECG as waveforms.

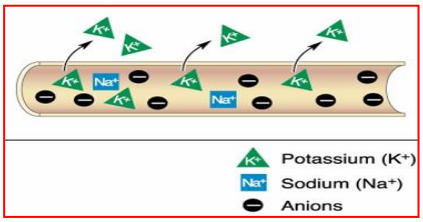
Depolarization occurs when the electric charge on the inside of the cell becomes positive and the electric ion charge on the outside of the cell becomes negative. In a healthy heart, depolarization (electrical activity) is followed by myocardial contraction (mechanical activity). It is possible to view electrical activity on the cardiac monitor, yet the patient may not have a palpable pulse.



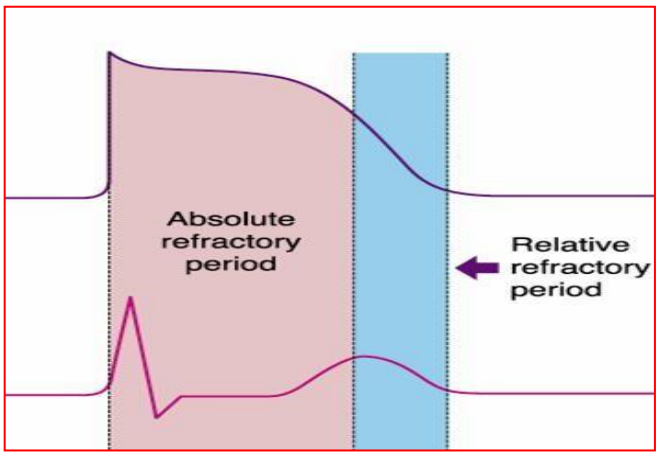


Repolarization takes place when the electrical charge returns to what it was prior to depolarization: negative ion charge on the inside and positive ion charge on the outside of the cell. Repolarization (electrical activity) is followed by myocardial relaxation (mechanical activity).





Refractory Period Once a cardiac cell is electrically excited, there follows a period of time during which the cell is either unable to respond to another stimulus (Absolute refractory period) or will only respond if the stimulus is stronger than normal (relative refractory period).



PROPERTIES OF CARDIAC TISSUE

Cardiac tissue has four main properties:

1. Automaticity Automaticity of cardiac tissue is the ability to produce or initiate spontaneous electrical impulses. The heart can initiate and maintain rhythmic activity without autonomic nervous system innervation. Impulses begin in the SA node pacemaker tissue, the primary pacemaker of the heart, and travel through all conduction pathways. More than one site within the heart has the ability to generate impulses and act as pacemakers, namely, the SA Node, the AV Node and the Purkinje Fibers. These sites have a slower intrinsic rate and will not initiate an impulse unless the SA Node fails. When the SA Node fails, the pacemaker site with the next highest intrinsic rate will take over.

2. Excitability Excitability of cardiac tissue is the ability to respond to a given stimulus. In the normal heart, the degree of excitability varies within the different periods of the cardiac cycle, the intensity and duration of the stimulus and with the degree of refractoriness of the myocardium at the time

the impulse is received. Each cardiac cell that receives an electrical impulse will alter its ionic composition and polarity. Once electrical stimulation begins in the cardiac cell it will continue until the entire cell is depolarized**.**

3. Conductivity Conductivity of cardiac tissue is the ability to conduct or transmit electrical impulses. A cardiac cell transfers an impulse to a neighboring cell very rapidly, so that all areas of the heart appear to depolarize simultaneously. The velocity of transmission varies in different cardiac tissues. Electrical impulses activate and depolarize cardiac tissue in a definite sequence. Impulses travel through the heart from right to left and from top to bottom.

4. Contractility Contractility of cardiac tissue is the ability of cardiac cell to shorten, causing cardiac muscle contraction in response to an electrical stimulus. Contractility can be enhanced through the use of cretin medications, like Digitalis, Dopamine, and Epinephrine.

How the Autonomic Nervous System affects cardiac Electrophysiology

Although they generate electrical impulses independently, myocardial cells are affected by both the sympathetic and parasympathetic divisions of the autonomic nervous system.

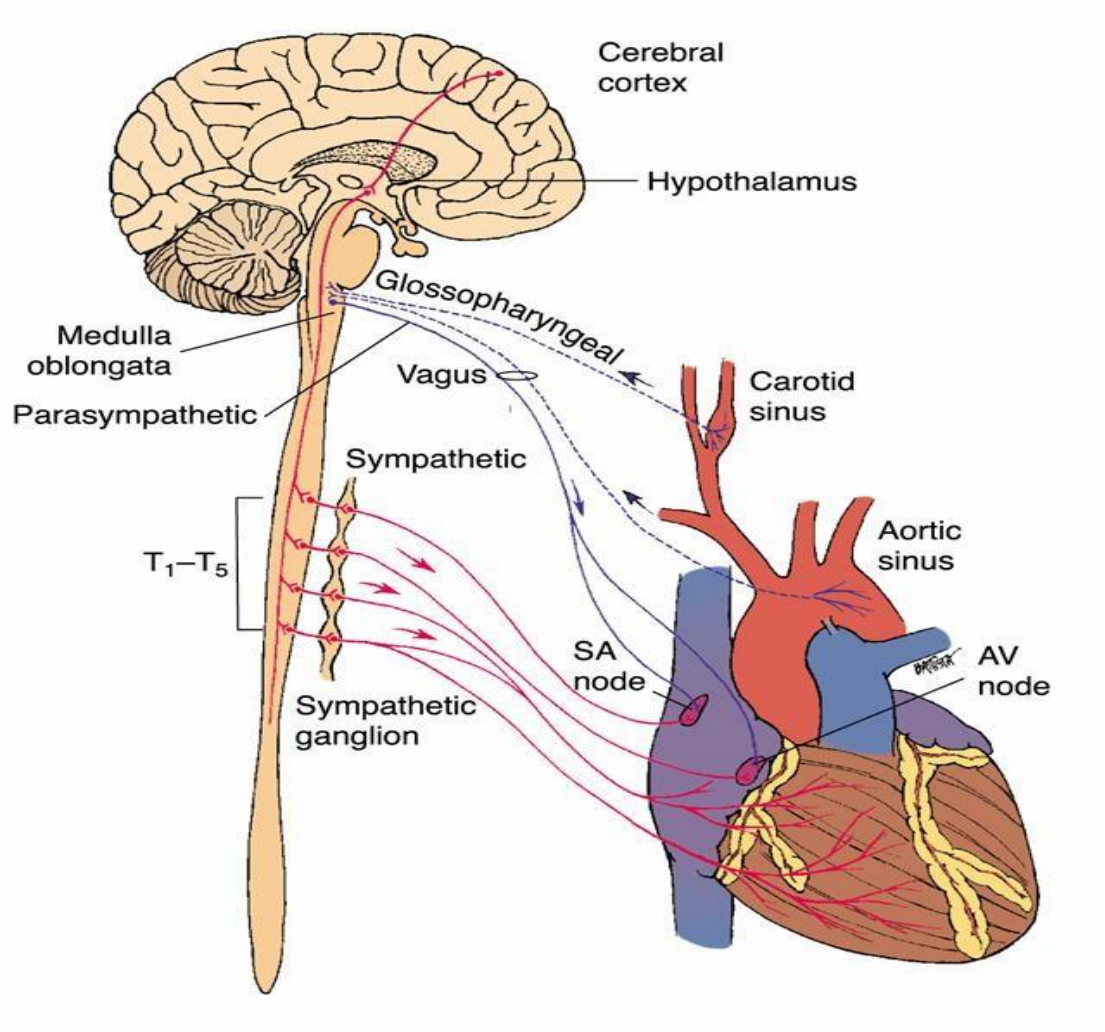
The Sympathetic Nervous System (SNS) Sympathetic Nervous System fibers extend to the entire conduction system and myocardium. Innervation of the sympathetic fibers enhances automaticity with the dominant influence exhibited within the ventricles. Stimulation of the SNS fibers will:

1. Increase heart rate
2. Increase speed of conduction through the AV Node
3. Increase force of ventricular contraction

The Parasympathetic Nervous System (PNS)

Parasympathetic Nervous System fibers, primarily via the vagus nerve, extend to the SA Node, atrial musculature and AV Node. Innervation of the parasympathetic fibers suppresses automaticity with the dominant influence exhibited within the atria. Stimulation of these fibers will:

1. Decrease heart rate
2. Decrease speed of conduction through the AV Node
3. Decrease force of atrial contraction



Autonomic nervous system innervation of the heart. (Aehlert, B, 1995)

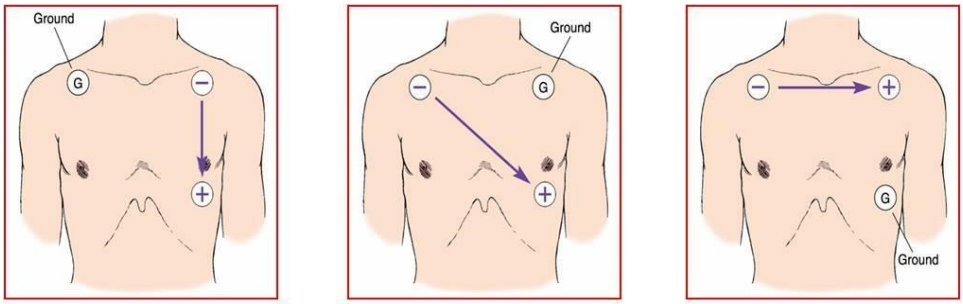
CARDIAC MONITORING

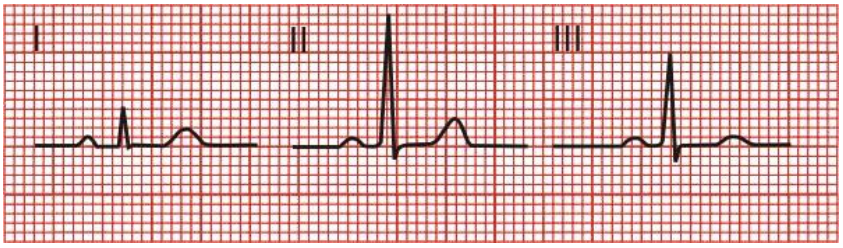
EKG Lead Placement

The lead position chosen for continuous bedside monitoring depends on unit policies and procedures. Even though Lead II is the most common lead in which continuous monitoring occurs, it is not considered to be ideal for the best visualization of both atrial and ventricular electrical activity. The best lead for continuous monitoring is considered to be Modified Chest Lead – I (MCL-I), also called Modified V-1, since this lead provides a view of the heart which clearly records the sequence of ventricular depolarization and repolarization and differentiates between right and left sided electrical activity.

Einthoven's Triangle

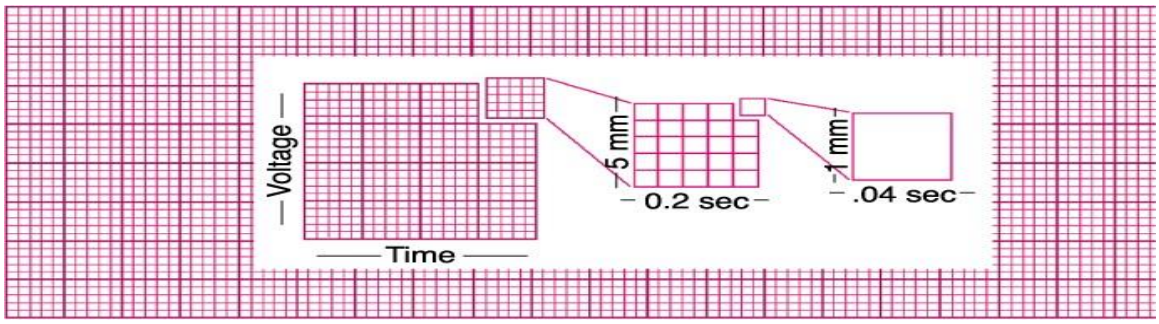
Einthoven's Triangle is the equilateral triangle formed by the electrode connections of the bipolar leads I, II, and III. Bipolar leads measure voltage between two extremities. The ground lead may be located in various positions. Normal depolarization travels from right to left and from the top to the bottom of the heart. Bedside monitors record positive waveforms when the electrical activity of depolarization moves toward the positive electrode and negative waveforms when the electrical activity moves away from the positive electrode.





ELECTROCARDIOGRAPHIC (ECG) PAPER

The ECG graph paper, made up of small and large squares, records cardiac electrical activity with regard to time and voltage. Time is represented on the horizontal axis as seconds and voltage on the vertical axis as millimeters or millivolts.



The Small Squares (Small Boxes)

1. Each small square equals 0.04 seconds
2. There are five small squares in one large square
3. 1500 small squares equals one minute (1500 x 0.04 = 60 seconds)
4. 75 small squares equals three seconds (75 x 0.04 = 3 seconds)
5. 150 small squares equals six seconds (150 x 0.04 = 6 seconds)

The Large Squares (Large Boxes)

1. Each large square equals 0.20 seconds
2. Each large square contains five small squares
3. 300 large squares equals one minute (300 x 0.20 = 60 seconds)
4. 15 large squares equals three seconds (15 x 0.20 = 3 seconds)
5. 30 large squares equals six seconds (30 x 0.20 = 6 seconds)

COMPONENTS OF THE NORMAL CARDIAC ELECTRICAL IMPULSE

P Waves

Represents the atrial depolarization

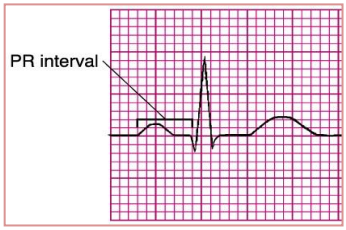
-
- Smooth and rounded
- Usually no more than 2.5 mm in height and 0.11 second in duration
- Should be upright in all lead except aVR
- May be descried notched, peaked, inverted, or may not be present.

PR Interval

Represents the atrial depolarization and repolarization of the atria

The P wave plus the PR segment equals the PR interval

- Begins with the onset of the P wave and ends with the onset of the QRS complex



- A normal PR interval indicates the electrical impulse was conducted normally through the atria, AV node, Bundle of His, Bundle branches, and Purkinje Fibers.
- Normally measures 0.12 to 0.20 second in adults; may be shorter in children and longer in older persons
- Normally shortens as heart rate increases

The PR interval is measured by counting the small squares and multiplying the number by 0.04.

QRS Complex

Represents ventricular depolarization

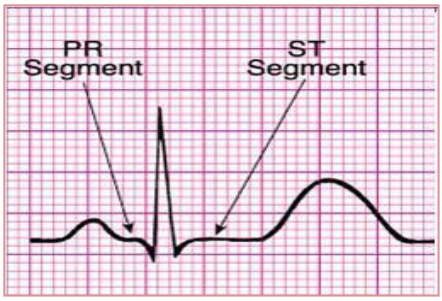
- Normal duration 0.04-0.12 seconds
- Measured from the beginning of the QRS complex to the end of the S
- q = first downward deflection before the R wave
- R = first initial upward deflection following the P wave
- s = first downward deflection following the R wav

The QRS interval (duration) measured by counting the small squares and multiplying the number by 0.04.

ST Segment

Represents ventricular depolarization

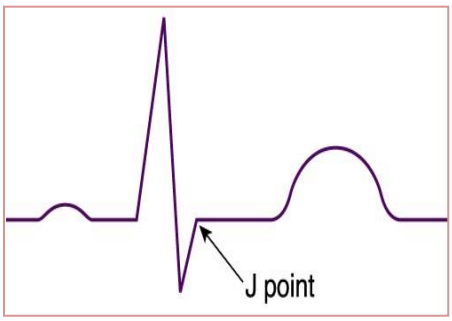
- Period between the completion of ventricular depolarization and beginning of final ventricular repolarization.
- Normally found on the isoelectric line; may be elevated or depressed(usually less than 1 mm)



J point

Represents the point at which the QRS complex and the ST segment met.

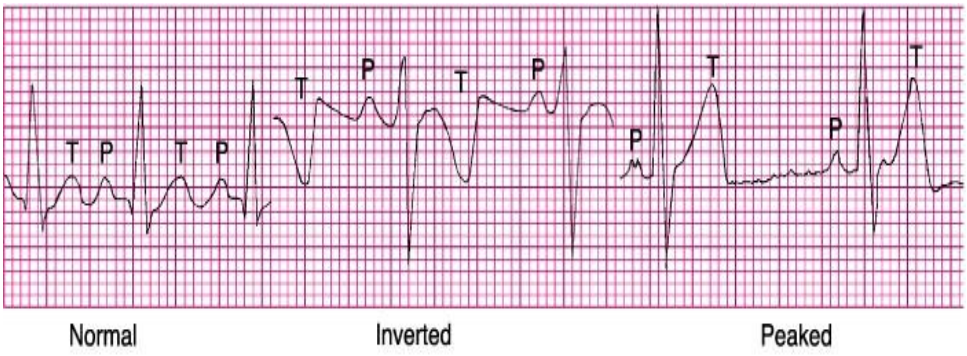
- Normally isoelectric



T Wave

Represents ventricular repolarization (recovery phase)

- Normally upright in all leads except aVR



QT Interval

Measured from the beginning of the QRS complex to the end of the T wave.

- Normal interval is dependent on rate and gender (normal 0.35-0.45 seconds) QT interval measured by counting the small squares and multiplying the number by 0.04.

Correlated or Corrected QT

- QTc is the QT interval correlated to HR
- QTc calculated by dividing QT by square root of R-R interval

$$QT_c = \frac{QT}{\sqrt{RR}}$$

U Wave

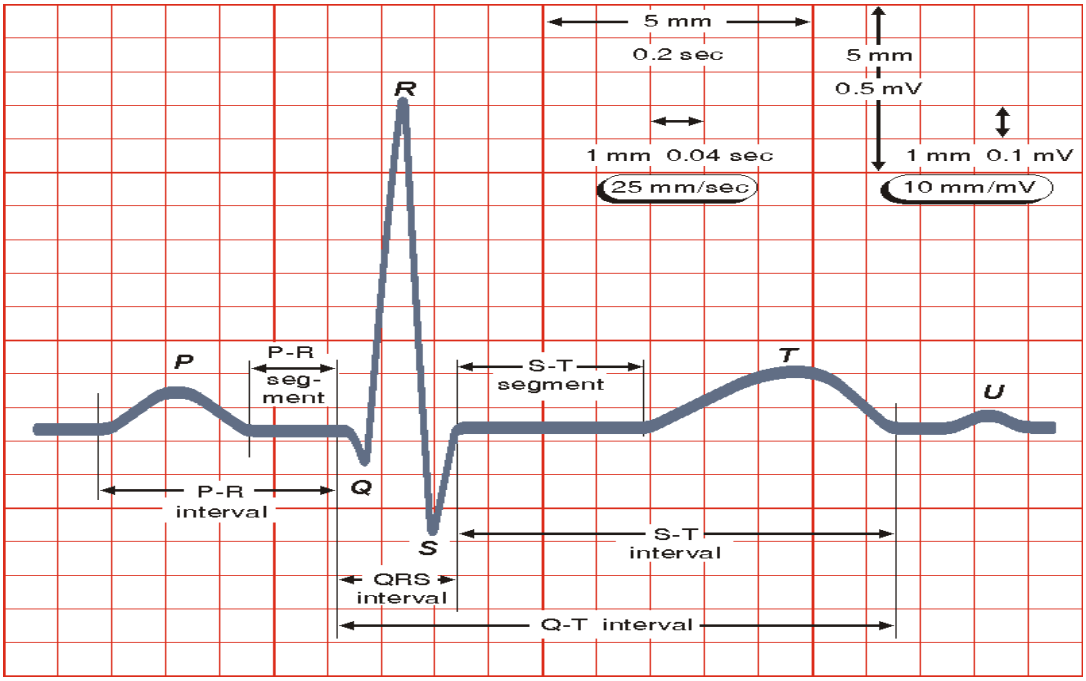
Thought to represent repolarization of the Purkinje fibers, but the significance is not definitely known. Not easily identified due to its low amplitude

- Rounded and symmetrical
- Usually less than 2 mm in amplitude
- In general, a U wave of more than 1.5 mm in height in any lead is considered abnormal

Abnormal U Waves

1. Abnormally tall U waves may be the result of:
   - Electrolyte imbalance
   - Medications
   - Hyperthyroidism
   - Central nervous system disease
   - Long QT syndrome

2. Negative U waves
   - Strongly suggestive of organic heart disease
   - May be seen in patients with ischemic heart disease



Components of the normal cardiac electrical impulse

STEP BY STEP RHYTHM ANALYSIS

To identify a rhythm accurately, learn to systematically analyze the cardiac rhythm by establishing a consistent approach to interpretation. There are different approaches to ECG analysis. The 6-step method is used in this self-study module.

STEP 1 **Assess the Rate**

A. Determine ventricular rate (R-R intervals)

B. Determine atrial rate (P-P intervals)

STEP 2 **Assess Rhythm/Regularity**

A.Ventricular Rhythm

a. Measure the distance between two consecutive R-R intervals

b. Compare with other R-R intervals

B.Atrial Rhythm

a. Measure the distance between two consecutive P-P intervals

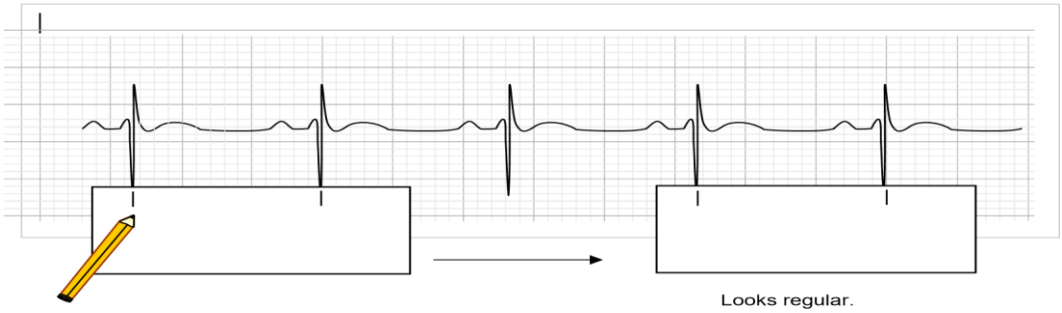
b. Compare with other P-P intervals

Determining the Rhythm

A. Caliper Method

B. Paper and Pencil Method

"Paper Calipers"



STEP 3 **Identify & Examine P Waves** Normally:

A. One P wave precedes each QRS complex

B. P waves occur regularly and appear similar in size, shape, and position

STEP 4 **PR Interval (PRI)**

A. Normal values range between 0.12 to 0.20 sec, if it longer it is prolonged

B. If the PR intervals are the same, they are said to be constant

C. If the PR intervals are different, is there a pattern?

a. Lengthening

b. Variable (no pattern)

STEP 5 **QRS Complex**

A.Presence and morphology

a. Narrow (normal) if it measures 0.12 second or less

b. Wide if it measures more than 0.12 second

STEP 6 **QT Interval &QTc Calculation**

A. Normal

B. Prolonged

DETERMINING HEART RATE FROM THE RHYTHM STRIP

There are four methods to calculate the heart rates:

1. Count the number of R waves in a six (6) seconds strip (6 inches = 6 seconds).

   Multiply the number of R waves by ten (10) to get a rhythm rate for one (1) minute. May be used for regular and irregular rhythms. Fastest but least accurate.

2. Count the number of large squares between two (2) R waves. Divide 300 by that number.

   Best used if the rhythm is regular.

3. Count the number of small squares between two (2) R waves. Divide 1500 by that number. Most accurate (preferred).

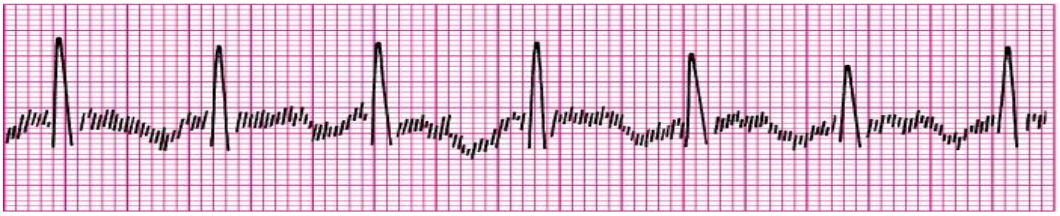
4. Sequence method

R-R interval (# large squares)	R-R interval in seconds	Heart rate per minute
1	0.2	300
2	0.4	150
3	0.6	100
4	0.8	75
5	1.0	60

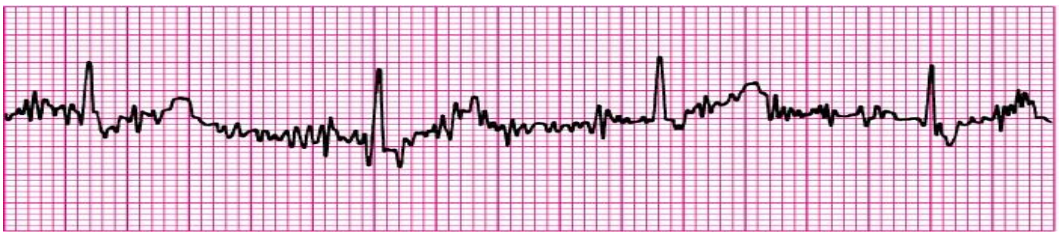
CARDIAC RHYTHM ARTIFACT

There are 4 types of artifact:

1. 60 Cycle Interference
   Causes:Electrical current leakage from equipment that is picked up by the ECG cable or electrodes. Also, the effect of other equipment plugged to the same outlet.



2. Patient Movement/Muscle Activity
   Causes:The patient is moving causing cable and electrode movement. There could be poor electrode contact with the skin.



3. Artifact-Mimicking Ventricular Arrhythmia
   Causes: Usually the cable is being moved in a very forceful manner such as whipping motions.

4. Wandering Baseline Artifact
   Causes:Usually related to the patient with a large chest. The electrodes on the chest are moving up and down with respirations.

Troubleshooting Artifact:

1. Check for crossing of lead wire such as monitor wires and other electrical equipment (i.e. call bell, transducer cables, ventilator tubing).

2. Check if monitor cable is in contact with electrical equipment (i.e. metal positions of ventilators).

3. If not in use, consider unplugging non-essential electrical equipment in contact with the patient.

4. Check for protruding wires from the ECG cable. If present, change cable.
5. Check to see if patient is moving (he/she could be having a seizure or is shivering).

6. Prevent excessive cable movement by attaching cable in a stress-prevention loop to patient or bed linen.

7. Ask patient to hold still momentarily. If artifact continues, check electrode position.

8. Move electrodes as far as possible to the periphery of the chest, away from the diaphragm and the chest movement.

SINUS RHYTHMS

NORMAL SINUS RHYTHM (NSR)

General Information

A. Impulse originates in the SA node

B. Intrinsic rate is 60-100 beats per minute

C. Conduction of the impulse is through the normal conduction pathways

D. Also referred to as Regular Sinus Rhythm (RSR)

Normal Sinus Rhythm			
Atrial Activity		Ventricular Activity	
P WAVE	Before each QRS	QRS	Present
MORPHOLOGY	Normal	MORPHOLOGY	Normal
PR INTERVAL	0.12 – 0.20 seconds	QRS INTERVAL	0.04 - 0.12 seconds
P-P RATE	60 - 100 bpm	R-R RATE	60-100 bpm
RHYTHM	Regular	RHYTHM	Regular

Clinical Significance

A. Normal cardiac function

B. Normal cardiac output

C. Adequate tissue perfusion

Treatment

A.None indicated

SINUS BRADYCARDIA (SB)

General Information

A. Impulse originates in the SA node

B. Heart rate is 40-60 beats per minute

C. Conduction of the impulse is through the normal conduction pathway

Sinus Bradycardia			
Atrial Activity		Ventricular Activity	
P WAVE	Before each QRS	QRS	Present
MORPHOLOGY	Normal	MORPHOLOGY	Normal
PR INTERVAL	0.12 - 0.20 seconds	QRS INTERVAL	0.04 - 0.12 seconds
P-P RATE	40 – 60 bpm	R-R RATE	40-60 bpm
RHYTHM	Regular	RHYTHM	Regular

Clinical Significance

A. Common in healthy young adults and the elderly, especially during sleeping hours

B. Common in early stage of myocardial infarction

C. Symptomatic bradycardia (reduced cardiac output)
1. Hypotension
2. Dizziness & Fainting
3. Low urine output
4. Chest pain

Treatment: Depends on heart rate and patient tolerance.

Goal: To increase automaticity of the SA node.

A. Drug Therapy
   a.Atropine b. Dopamine infusion c. Epinephrine infusion

B. Cardiac pacing

SINUS TACHYCARDIA (ST)

General Information

A. Impulse originates in the SA node

B. Intrinsic rate is 100-150 beats per minute

C. Conduction of the impulse is through the normal conduction pathways

Sinus Tachycardia			
Atrial Activity		Ventricular Activity	
P WAVE	Before each QRS	QRS	Present
MORPHOLOGY	Normal	MORPHOLOGY	Normal
PR INTERVAL	0.12 – 0.20 seconds	QRS INTERVAL	0.04 - 0.12 seconds
P-P RATE	100-150 bpm	R-R RATE	100-150 bpm
RHYTHM	Regular	RHYTHM	Regular

Clinical Significance:

A. May be caused by:
   1. Anxiety & Pain
   2. Fever

3. Exercise
4. Hyperthyroidism
5. Shock
6. Blood loss

B. May occur after drinking coffee, tea or alcohol

C. May occur as a side effect to sympathomimetic drugs

Treatment: Depends on heart rate and patient tolerance.

Goal: To maintain cardiac output and decrease myocardial O2 consumption by decreasing heart rate.

A. If stable: treat the underlying cause: a. Anti-pyretic, fluids, analgesia, etc..

B. If BP dropped: give fluids and treat the underlying cause.

SINUS ARRHYTHMIA (SA)

General Information

A. Impulse originates in the SA node

B. There is a cyclic fluctuation in the rate

C. The SA node and lungs are both innervated by the parasympathetic nervous system (PNS) via the vagus nerve. The SA node may be affected by respirations, and sinoatrial discharge may gradually increase with inspirations and gradually decrease with expirations. If these effects are marked, the overall rhythm will be irregular

D. The ventricular rate further describes the rhythm

Sinus Arrhythmia	
Atrial Activity	Ventricular Activity

P WAVE	Before each QRS	QRS	Present
MORPHOLOGY	Normal	MORPHOLOGY	Normal
PR INTERVAL	0.12 – 0.20 seconds	QRS INTERVAL	0.04 - 0.12 seconds
P-P RATE	60-100 bpm	R-R RATE	60-100 bpm
RHYTHM	Increase during inspiration; slows during expiration	RHYTHM	Increase during inspiration; slows during expiration

Clinical Significance

A. Considered a normal physiological process in children and young adults

B. May occur with digitalis toxicity and increased intracranial pressure

C. A non-respiratory form may occur with heart disease and when the patient is bradycardic

Treatment: Depends on heart rate and patient tolerance.

Goal:A.To maintain cardiac output.

B.Treat the underlying cause

SINOATRIAL BLOCK (SA BLOCK)

General Information

A. Also referred to as Sinoatrial Arrest

B. SA Block occurs because conduction is depressed between the SA node and the atria. When this happens, the normal sinus impulse is formed but fails to reach the atria. As a result, the atria and the ventricles are not depolarized

C. One QRST complex is missing

D. This should be considered an “event”. It alone is not a rhythm

Sinoatrial Block			
Atrial Activity		Ventricular Activity	
P WAVE	Present except for failed P wave	QRS	Present except for failed QRST
MORPHOLOGY	Normal	MORPHOLOGY	Normal
PR INTERVAL	Underlying rhythm	QRS INTERVAL	0.04 - 0.12 seconds
P-P RATE	Underlying rhythm	R-R RATE	Underlying rhythm
RHYTHM	Irregular	RHYTHM	Irregular

Clinical Significance

A. May be a result of drug therapy
   a. Quinidine
   b. Digoxin
   c. Potassium

B. The severity of the symptoms depends upon the number and frequency of SA Blocks. SA Blocks a decrease in cardiac output which results in diminished blood pressure

C. SA Blocks may result in a reduction of blood supply to the SA node from the coronary arteries after a myocardial infraction.

Treatment: Depends on length of ventricular asystole and patient tolerance.

Goal: To enhance electrical conduction.

A. Drug therapy:
   a.Atropine b. Dopamine infusion c. Epinephrine infusion

B. Cardiac Pacing

C. If no pulse start CPR

ATRIAL ARRHYTHMIAS

PREMATURE ATRIAL CONTRACTION (PAC)

General Information

A. Impulse originates in atrial tissue, other than the SA node.

B. The PAC appears like the preceding sinus beat. It should be considered as an "event". It alone is not a rhythm.

C. PACs may occur alone (isolated) or occur in patterns:

Every other beat = bigeminy
Every third beat = trigeminy
Every fourth beat = quadrigeminy
Two together = pair or couplet
Three together = runs of atrial tachycardia

Premature Atrial Contractions			
Atrial Activity		Ventricular Activity	
P WAVE	before each QRS	QRS	Present
MORPHOLOGY	Changes	MORPHOLOGY	may vary from underlying rhythm
PR INTERVAL	Varies	QRS INTERVAL	0.04 - 0.12 seconds
P-P RATE	underlying rhythm	R-R RATE	underlying rhythm
RHYTHM	underlying rhythm	RHYTHM	underlying rhythm

Clinical Significance

A. Occurs in rheumatic heart disease (RHD)

B. Isolated PACs may occur in apparently normal persons

C. May be caused by hyperirritability of atrial muscle by:
   a. Drug therapy
   b. Anxiety
   c. Caffeine
   d. Diminished myocardial oxygenation

D. Frequent PACs forewarn of, and possibly initiate, other more serious atrial dysrhythmias, such as: a.Atrial Tachycardia
   b. Atrial Flutter
   c. Atrial Fibrillation

Treatment: Depends on the frequency of PACs.

Goal: To decrease automaticity.

A. Omit stimulants
B. Check and correct electrolytes
C. Drug therapy may be required if PACs are frequent : Amiodarone

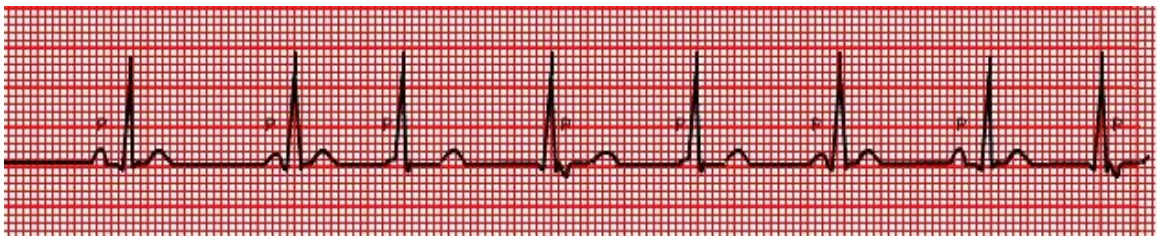
WANDERING ATRIAL PACEMAKER (WAP)

General Information

A. The impulse site moves between the SA node, the atrial conduction tissue, irritable atrial foci and may even shift to the AV junction

B. If the lower pacemaker (AV node) gains control there will be a change in the P wave morphology (shape). This may be accompanied by changes in the P-P interval, the RR interval and the PR interval. The PR interval may change from being absent to being greater than 0.20 seconds. When the PR interval is less than 0.12 seconds, or if the P wave is missing, those beats are Junctional in origin. The PR interval may be more than 0.20 seconds if First Degree AV Block (see section 6 on atrioventricular blocks) is present

C. The rhythm becomes cyclic much like Sinus Arrhythmia. The difference is that in Sinus Arrhythmia, the pacemaker does not change, therefore the PR interval remains constant and the P wave morphology is unchanged



Wandering Atrial Pacemaker			
Atrial Activity		Ventricular Activity	
P WAVE	Before each QRS, or absent	QRS	Present
MORPHOLOGY	Changes	MORPHOLOGY	Normal
PR INTERVAL	Varies	QRS INTERVAL	0.04 - 0.12 seconds
P-P RATE	Usually 60-100 bpm (varies if lower pacemaker takes over)	R-R RATE	Usually 60-100 bpm (varies if lower pacemaker takes over)
RHYTHM	Regular/irregular	RHYTHM	Regular/irregular

Clinical Significance

A. The SA node may be failing

B. There may be a reduction in ventricular rate

C. May be related to underlying heart disease (i.e. post myocardial infarction)

Treatment: Depends on heart rate and patient tolerance.

Goal: To increase automaticity of the SA node and increase conduction, and maintain cardiac output.

A. Drug Therapy
   a.Atropine b. Dopamine infusion c. Epinephrine infusion

B. Cardiac Pacing

MULTIFOCAL ATRIAL TACHYCARDIA (MAT)

General Information

A. MAT is basically WAP with a faster rate

B. The term multifocal atrial tachycardia is used when atrial impulses show at least three different P wave morphologies and a ventricular rate of 100 or greater.

C. Ventricular response during multifocal atrial tachycardia is often rapid

Multifocal Atrial Tachycardia (MAT)			
Atrial Activity		Ventricular Activity	
P WAVE	Before each QRS, or absent	QRS	Present
MORPHOLOGY	Changes	MORPHOLOGY	Normal
PR INTERVAL	Varies	QRS INTERVAL	0.04 - 0.12 seconds
P-P RATE	Above 100 bpm	R-R RATE	Above 100 bpm
RHYTHM	Regular/irregular	RHYTHM	Regular/irregular

Clinical Significance

A. Associated with chronic obstructive lung disease or significant atrial disease

B. Multifocal atrial tachycardia is often a precursor of atrial fibrillation and on physical examination may actually be confused with atrial fibrillation, since an irregularly irregular ventricular response is noted

D.May be related to underlying heart disease (i.e. post myocardial infarction)

Goal: To maintain cardiac output

Treatment:

A. Vagal Stimuli - Ice on forehead/bridge of nose for 10 sec

B. Drug Therapy : B. Blockers, C. Channel Blocker, Amiodarone

ATRIAL TACHYCARDIA (ATRIAL TACH OR AT)

General Information

A. This is an atrial ectopic rhythm. The irritable focus originates in the atrial tissue

B. Atrial tachycardia is defined as three (3) or more consecutive PACs

C. The atrial rate ranges from 150-250 per minute

D. Normally, all atrial impulses will go to the ventricles, producing the same ventricular rate as the atrial rate

Atrial Tachycardia			
Atrial Activity		Ventricular Activity	
P WAVE	Before each QRS	QRS	Present
MORPHOLOGY	buried in preceding T wave	MORPHOLOGY	May be different from QRS of the underlying rhythm
PR INTERVAL	Varies	QRS INTERVAL	0.04 - 0.12 seconds, or less
P-P RATE	150-250 bpm	R-R RATE	150-250 bpm
RHYTHM	Regular	RHYTHM	Regular

Clinical Significance

A. Associated with rheumatic heart disease and myocardial infraction

B. May be associated with the sudden withdrawal of cardiac drugs

C. Rapid ventricular rates cause a decrease in cardiac output, resulting in:
   a. Hypotension
   b. Palpitations
   c. Chest pain
   d. Congestive heart failure

Treatment: Depends on the frequency of PACs.

Goal: To decrease automaticity and restore cardiac output

A. Vagal maneuvers: Ice on forehead/bridge of nose for 10sec

B. Drug Therapy : B. Blockers, C. Channel Blocker, Amiodaron

SUPRA VENTRICULAR TACHYCARDIA (SVT)

General Information

A. SVT is a term used for narrow QRS complex tachycardias that are known to originate above the ventricles, but whose exact mechanism is unknown. The rate is between 140300 bpm.

B. The term SVT may also be used for all tachyarrhythmias that originate above the bifurcation of the bundle of His
   a. Sinus tachycardia
   b. Atrial tachycardia
   c. Atrial flutter
   d. Atrial fibrillation
   e. Junctional tachycardia

C. If P waves are visible, the atrial rate usually allows one to know the mechanism of the arrhythmia i.e. Atrial tachycardia: 150-250, Atrial flutter: 250-350, etc.

D. If no P waves are visible, the term SVT is used, since without P waves it is not possible to know for sure where the dysrhythmia originates.

Supra Ventricular Tachycardia (SVT)			
Atrial Activity		Ventricular Activity	
P WAVE	Usually not visible	QRS COMPLEX	Present
MORPHOLOGY	n/a	MORPHOLOGY	Normal
RHYTHM	n/a	RHYTHM	Regular
P-P RATE	n/a	R-R RATE	140-300 bpm
PR INTERVAL	n/a	QRS INTERVAL	0.04-0.12 seconds

Clinical Significance

A. Can be caused by MI, congestive cardiac failure, hypoxia, or electrolyte disturbances

B. Usually well tolerated and often paroxysmal in nature.

C. Can sometimes be easily terminated by a vagal maneuver.

D. If rate is very rapid and sustained, cardiac output may become compromised and symptoms will occur.

Treatment*:* Depends on duration of rapid ventricular rate and patient tolerance

Goal*:* To restore normal heart rate, cardiac output, and tissue perfusion.

A. If <u>stable</u>

a. Vagal maneuvers i.e. Ice on forehead/bridge of nose 10sec

b. Adenosine

c. Ca+2 channel blockers

d. Beta blockers

e. Amiodarone

B. Correct electrolytes (if imbalanced)

C. If unstable or unresponsive to drug therapy - Synchronized Cardioversion

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA (PSVT)

General Information

A. This is an atrial ectopic rhythm. The irritable focus originates in the atrial tissue

B. PSVT differs from SVT only by its sudden onset and cessation

C. The atrial rate ranges from 150-250 per minute

D. Normally, all atrial impulses will go to the ventricles, producing the same ventricular rate as the atrial rate

Paroxysmal Supraventricular Tachycardia			
Atrial Activity		Ventricular Activity	
P WAVE	Before each QRS	QRS	Present
MORPHOLOGY	Buried in preceding T wave	MORPHOLOGY	May be different from QRS of the underlying rhythm
PR INTERVAL	Usually unmeasurable	QRS INTERVAL	0.04 - 0.12 seconds, or less
P-P RATE	150-250 bpm	R-R RATE	150-250 bpm
RHYTHM	Regular	RHYTHM	Regular

Clinical Significance

A. May be caused by:
   a. Myocardial infarction
   b. Congenital heart disease
   c. Chronic pulmonary disease
   d. Hypertension
   e. Cardiomyopathy
   f. Hyperthyroidism

B. Rapid ventricular rates cause a decrease in cardiac output, resulting in:
   a. Hypotension
   b. Palpitations
   c. Chest pain
   d. Congestive heart failure

Treatment: Depends on duration of rapid ventricular rate and patient tolerance

Goal: To decrease automaticity and conductivity, and to improve cardiac output.

A. If stable
   a. Vagal maneuvers i.e. Ice on forehead/bridge of nose 10sec
   b. Adenosine
   c. Ca+2 channel blockers
   d. Beta blockers
   e. Amiodarone

B. Correct electrolytes (if imbalanced)

C. If unstable or unresponsive to drug therapy - Synchronized Cardioversion

ATRIAL FLUTTER (A FLUTTER)

General Information

A. This is an atrial ectopic rhythm. The irritable focus originates in the atrial tissue and becomes dominant

B. The atrial rate is regular and ranges from 250-350 bpm

C. The atrial electrical activity appears as saw-toothed Flutter waves ("F" waves) on the ECG tracing. P waves are replaced by these "F" waves

D. The ventricular rhythm is regular or irregular. Usually the ventricular rate is 60-100 bpm, depending on how the atrial activity is conducted through the AV junction to the ventricles. The ventricular rate may be higher, especially during acute episodes

E. The atrial impulse either travels in a "circuitous" movement or is from a single ectopic focus discharging at a rapid rate

Atrial Flutter			
Atrial Activity		Ventricular Activity	
P WAVE	Refered to as "F" waves	QRS	present
MORPHOLOGY	sawtooth shaped	MORPHOLOGY	normal
PR INTERVAL	None	QRS INTERVAL	0.04 - 0.12 seconds
F-F RATE	250-350 bpm	R-R RATE	varies with atrial conduction
RHYTHM	Regular	RHYTHM	regular/irregular

Clinical Significance

A. May be paroxysmal or chronic

B. Common in:
   a. Rheumatic heart disease
   b. Coronary artery disease
   c. Hypertensive heart disease

C. Increased ventricular rates cause decreased cardiac output:
   a. Hypotension
   b. Palpitations
   c. Chest pain

d. Congestive heart failure

D. Stasis of blood in the atria increases the potential for clot formation, known as mural thrombi

Treatment: Depends on ventricular rate and patient tolerance.

Goal: To decrease rapid ventricular rate and maintain cardiac output. A.If Stable,

Drug therapy :

a. Digoxin
b. Ca+2 channel blockers , Beta blockers
c. Amiodarone
d. Anticoagulants : Heparin/Warfarin

B.If Unstable : Synchronized Cardioversion

	Duration < 48 hrs	**Duration > 48 hrs**
Control Rate	β blockers Ca^{2+} Channel blockers	β blockers Ca^{2+} Channel blockers
Control Rhythm	Syncronized Cardioversion Amiodarone	Anticoagulate for 3 weeks, then Synchronized Cardioversion, then Anticoagulate for another 4 weeks

Table.1

ATRIAL FIBRILLATION (A FIB)

General Information

A. This is an atrial ectopic rhythm, and can be classified as a supraventricular rhythm.

B. There is no visible P wave. Atrial activity appears as fibrillatory waves on the ECG tracing

C. Atrial impulses bombard the AV node; the AV node selects and conducts these impulses randomly

D. The atrial activity determines the name of the rhythm. The atrial rate is 300-600 fibrillations per minute and is unmeasurable

E. The ventricular response to the atrial activity determines the rate and terminology of the rhythm. It is always irregular

Atrial Fibrillation			
Atrial Activity		Ventricular Activity	
P WAVE	Referred to as "f" waves	QRS	Present
MORPHOLOGY	undulating, wavy baseline	MORPHOLOGY	normal
PR INTERVAL	None	QRS INTERVAL	0.04 - 0.12 seconds
F-F RATE	300-600 and unmeasurable	R-R RATE	Varies
RHYTHM	Irregular	RHYTHM	Irregularly irregular

Clinical Significance

A. May be acute or chronic

B. In uncontrolled atrial fibrillation, the cardiac output may drop, causing:
   a. Hypotension
   b. Palpitations
   c. Chest pain

C. Common in
   a. Congestive heart failure
   b. Coronary artery disease
   c. Rheumatic heart disease
   d. Hypertensive heart disease

D. Stasis of blood in the atria increases the potential for clot formation, known as mural thrombi

Treatment: Depends on ventricular rate and patient tolerance.

Goal: To increase or decrease automaticity and conductivity.

A. <u>If Stable,</u> Drug therapy:
   a. Digoxin
   b. Ca+2 channel blockers , Beta blockers
   c. Amiodarone
   d. Anticoagulants : Heparin/Warfarin

B. <u>If unstable</u> : Synchronized Cardioversion

JUNCTIONAL ARRHYTTHMIAS

PREMATURE JUNCTIONAL CONTRACTION(PJC)

General Information

A. Impulse originates in Junctional tissue, and paces the heart for a single beat. These are *ectopic* beats

B. The P wave (if visible) of the PJC is always inverted, falling before, during or after the QRS complex. It should be considered as an "event", not a rhythm

C. PJCs may occur alone (isolated) or occur in patterns:
Every other beat = bigeminy
Every third beat = trigeminy
Every fourth beat = quadrigeminy

D. There usually is a noncompensatory pause which reflects retrograde conduction to the atria

E. The underlying rhythm is usually sinus or atrial in origin

Premature Junctional Contractions			
Atrial Activity		Ventricular Activity	
P WAVE	Present	QRS	Present
MORPHOLOGY	Always inverted, falling before, during or after QRS complex	MORPHOLOGY	Normal
PR INTERVAL	Absent or shortened (less than 0.12 seconds)	QRS INTERVAL	0.04 - 0.12 seconds
P-P RATE	Underlying rhythm	R-R RATE	Underlying rhythm
RHYTHM	Underlying rhythm	RHYTHM	Underlying rhythm

Clinical Significance

A. Occurs in rheumatic heart disease (RHD) and acute MI

B. Unusual in healthy people

C. Hyperirritability of Junctional tissue due to digitalis toxicity or excessive caffeine intake

D. Frequent PJCs forewarn of, and possibly initiate, other more serious arrhythmias such as Junctional Tachycardia

Treatment: Depends on the frequency and patient's toleration of PJCs

.

Goal: To decrease automaticity.

A. If asymptomatic, usually no treatment is necessary

B. If symptomatic, treat the underlying cause. i.e. digitalis toxicity, electrolytes imbalanced

C. If PJCs is more frequent, Amiodarone can be given

JUNCTIONAL RHYTHM (JR)

General Information

A. This dysrhythmia originates in the AV junctional tissue. It can be classified as a Supraventricular rhythm

B. The atria are depolarized in a reverse direction. This is known as retrograde atrial depolarization, and is seen on the oscilloscope as P wave inversion

C. The P wave and PR interval appear in three (3) ways:

1. P wave is inverted and falls before the QRS complex. The PR interval is shortened.
2. P wave is inverted and falls within the QRS complex. There is no PR interval.
3. P wave is inverted and falls after the QRS complex. There is no PR interval.

Junctional Rhythm			
Atrial Activity		Ventricular Activity	
P WAVE	Present	QRS	Present
MORPHOLOGY	Always inverted, falling before, during or after QRS complex	MORPHOLOGY	Normal
PR INTERVAL	Absent or shortened	QRS INTERVAL	0.04 – 0.12 seconds
P-P RATE	40-60 bpm	R-R RATE	40-60 bpm
RHYTHM	Regular	RHYTHM	Regular

Treatment: Depends on heart rate and patient tolerance.

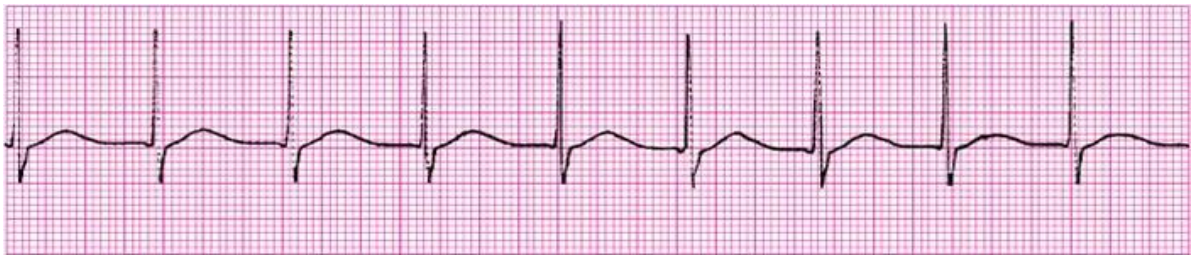
Goal:A.To increase automaticity of the SA node.

B. Drug Therapy

a.Atropine b. Dopamine infusion c. Epinephrine infusion

C.Cardiac pacing

ACCELERATED JUNCTIONAL RHYTHM (AJR)



Accelerated Junctional Rhythm			
Atrial Activity		Ventricular Activity	
P WAVE	Present	QRS	Present
MORPHOLOGY	Always inverted, falling before, during or after QRS complex	MORPHOLOGY	Normal
PR INTERVAL	Absent or shortened (less than 0.12 seconds)	QRS INTERVAL	0.04 - 0.12 seconds
P-P RATE	60-100 bpm	R-R RATE	60-100 bpm
RHYTHM	Regular	RHYTHM	Regular

Clinical Significance

A. With JR and AJR cardiac output may be reduced because the atrial kick is lost

B. May occur transiently in the early stages of myocardial infarction, as a result of open heart surgery, or inducted by drug toxicity

Treatment: Depends on ventricular rate and patient tolerance.

Goal: To increase or decrease automaticity.

A. Correct the Cause, Remove drug causing toxicity – Correct electrolytes
B. Atrial Cardiac Pacing

JUNCTIONAL TACHYCARDIA (JT)

Junctional Tachycardia			
Atrial Activity		Ventricular Activity	
P WAVE	Present	QRS	Present
MORPHOLOGY	Always inverted, falling before, during or after QRS complex	MORPHOLOGY	Normal
PR INTERVAL	Absent or shortened (less than 0.12 seconds)	QRS INTERVAL	0.04 - 0.12 seconds
P-P RATE	Greater than 100 bpm	R-R RATE	Greater than 100 bpm
RHYTHM	Regular	RHYTHM	Regular

Clinical Significance

A. Cardiac output may be reduced because the atrial kick is lost

B. May occur transiently in the early stages of myocardial infarction, as a result of open heart surgery, or induced by drug toxicity

Treatment: Depends on ventricular rate and patient tolerance.

Goal: To increase or decrease automaticity.

a. Remove drug causing toxicity

b. Correct electrolytes (if imbalanced)

c. Vagal maneuvers i.e. Ice on forehead/bridge of nose 10sec

ATRIOVENTRICULAR BLOCKS

FIRST DEGREE ATRIOVENTRICULAR BLOCK (1˚ AVB or 1˚ Heart Block)

General Information

A. Most common conduction disturbance

B. A prolonged PR interval is the only difference between sinus rhythms and First Degree AV Block

First Degree Atrioventricular Block			
Atrial Activity		Ventricular Activity	
P WAVE	Before each QRST	QRS	Present
MORPHOLOGY	Normal	MORPHOLOGY	Normal
PR INTERVAL	Greater than 0.20 seconds	QRS INTERVAL	0.06 - 0.12 seconds
P-P RATE	Underlying rhythm	R-R RATE	Underlying rhythm
RHYTHM	Regular	RHYTHM	Regular

Clinical Significance

A.May be due to:

a. Chronic degenerative changes in the elderly
b. Myocardial infarction
c. Coronary artery disease
d. Rheumatic heart disease

B.Drug toxicity:

a. Digoxin
b. Quinidine
c. Potassium
d. Ca+2 channel blockers
e. Beta blockers

Treatment: Depends on heart rate and patient tolerance.

Goal: To maintain cardiac output.

A. Drug therapy for slow ventricular response:
   a. Atropine
   b. Epinephrine Infusion
   c. Dopamine Infusion

B. Cardiac pacing

C. Treat the underlying cause

D. Observe closely for progression to higher degrees of block (2˚ AV Block and 3˚ AV Block)

SECOND DEGREE ATRIOVENTRICULAR BLOCK TYPE I or WENCKEBACH (2˚ AVB-TYPE I or 2˚ HEART BLOCK-TYPE I)

General Information

A. More P waves than QRS complexes

B. The P to P intervals are regular

C. The PR interval is progressively longer until a QRS is dropped

D. The R to R intervals are irregular and may be progressively shorter

E. The AV block occurs above the Common Bundle of His

F. The right coronary artery supplies this portion of the AV node

Second Degree Atrioventricular Block – Type I			
Atrial Activity		Ventricular Activity	
P WAVE	Present	QRS	Present
MORPHOLOGY	Normal	MORPHOLOGY	Normal
PR INTERVAL	is progressively prolonged until a P wave is not conducted	QRS INTERVAL	0.06 – 0.12 seconds
P-P RATE	regular	R-R RATE	varies, may get progressively shorter
RHYTHM	regular	RHYTHM	irregular

Clinical Significance

A. Common with acute myocardial infarction and infections of the heart

B. It is a transient rhythm

C. Drug toxicity:
   a. Digoxin
   b. Quinidine
   c. Pronestyl
   d. Ca+2 channel blocker
   e. Beta blockers

Treatment: Depends on heart rate and patient tolerance.

Goal: To improve conductivity and to maintain heart rate and cardiac output.

A. Drug Therapy
   a. Atropine
   b. Epinephrine Infusion
   c. Dopamine Infusion

B. Cardiac pacing

C. Hold drugs that cause toxicity

SECOND DEGREE ATRIOVENTRICULAR BLOCK TYPE II (2˚ AVB-TYPE II or 2˚ HEART BLOCK-TYPE II)

General Information

A. More P waves than QRS complexes

B. The PR interval is constant

C. QRS complexes are dropped at intervals

D. Blood supply is reduced to portions of the Common Bundle of His

Second Degree Atrioventricular Block – Type II			
Atrial Activity		Ventricular Activity	
P WAVE	Present	QRS	Present
MORPHOLOGY	Normal; more P waves than QRS complexes	MORPHOLOGY	Normal
PR INTERVAL	0.12 – 0.20 and remains constant until non-conducted P wave	QRS INTERVAL	0.04 - 0.12 seconds or greater
P-P RATE	60-100	R-R RATE	40 and greater
RHYTHM	Regular	RHYTHM	Irregular

Clinical Significance

A. This rhythm is a result of necrotic damage to the conduction system, common with anterior wall myocardial infarction and infections of the heart

B. It usually a transient rhythm, and may progress suddenly to 3˚ AV Block and asystole

Treatment: Depends on heart rate and patient tolerance.

Goal: To improve conductivity and to maintain heart rate and cardiac output.

A. Drug Therapy
   a. Atropine
   b. Epinephrine Infusion
   c. Dopamine Infusion

Cardiac pacing is the treatment of choice

THIRD DEGREE ATRIOVENTRICULAR BLOCK (3˚ AV BLOCK or COMPLETE HEART BLOCK or AV DISSOCIATION)

General Information

A. The atria beat independently of the ventricles

B. There is a complete block in the bundles, and no atrial impulses are activating the ventricles

C. The QRS complexes are
   a. *Narrow* when the electrical impulse is generated high in the ventricular conduction system, close to the AV node
   b. *Wide* when the electrical impulse is generated below in the ventricular conduction system, far from the AV node

D. The SA node usually fires at an intrinsic rate of 60-100 bpm and the ventricles beat at an intrinsic rate of 40 or less. The atrial activity will be faster than the ventricular rate and there will be more P waves than QRS complexes

E. The P-P intervals are regular. The R-R intervals are regular

Third Degree Atrioventricular Block			
Atrial Activity		Ventricular Activity	
P WAVE	Present	QRS	Present
MORPHOLOGY	Normal; more P waves than QRS complexes	MORPHOLOGY	Normal
PR INTERVAL	Variable	QRS INTERVAL	0.04 – 0.12 seconds or greater
P-P RATE	60-100 bpm	R-R RATE	40 or less
RHYTHM	Regular	RHYTHM	Regular

Clinical Significance

A. Common with acute myocardial infarction and degenerative disease in the elderly

B. Slow ventricular rate resulting from decreased cardiac output causes:
   a. Dizziness
   b. Fainting
   c. Weakness
   d. Hypotension

Treatment: is always indicated.

Goal: To improve conductivity and to maintain heart rate and cardiac output.

A. Drug Therapy
   a. Atropine
   b. Epinephrine Infusion
   c. Dopamine Infusion

B. Cardiac pacing is the treatment of choice

VENTRICULAR ARRHYTHMIAS

PREMATURE VENTRICULAR CONTRACTION (PVC)

General Information

A. Impulse may originate anywhere in the ventricular tissue. These are ectopic beats.

B. PVCs should be considered as an "event". It is not a rhythm.

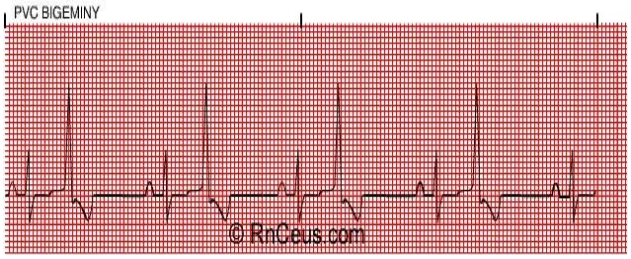
C. PVCs may occur alone (isolated) or occur in patterns:
Every other beat = bigeminy
Every third beat = trigeminy
Every fourth beat =quadrigeminy

D. The morphology of the QRS complex may be:
Uniformed: There is only one irritable area (or *focus*) in the ventricle.
Multiformed: There is more than one irritable area (or *foci*) in the ventricle. The complexes have different forms.

E. Classic characteristics of a PVC:
1. Occurs prematurely
2. QRS is greater than 0.12 seconds
3. The deflection of the QRS complex is opposite to the deflection of the ST segment and T wave
4. Is generally followed by a compensatory pause

PREMATURE VENTRICULAR CONTRACTIONS

PVC Bigemany

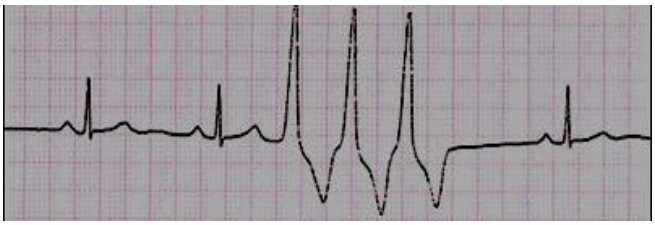


PVC Trigemany

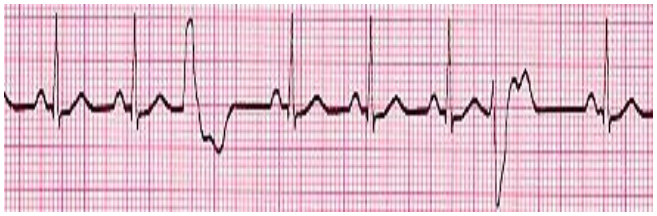
Pair or CoupletUniformed PVCs

Triplet

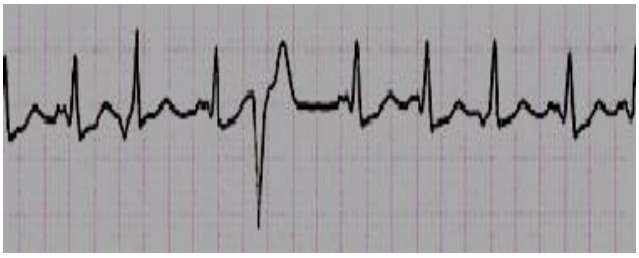
Multiformed PVCs

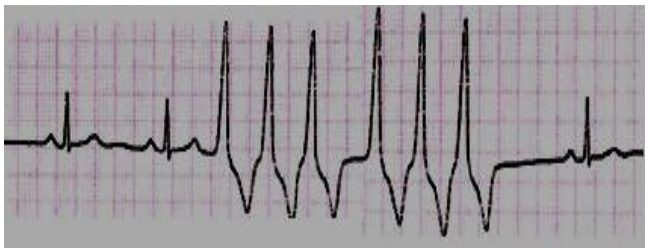


R-on-T Phenomena



Run of V Tach





Quadrigeminy PVCs

Premature Ventricular Contractions			
Atrial Activity		Ventricular Activity	
P WAVE	Present	QRS	Present
MORPHOLOGY	Buried in or following QRS complex	MORPHOLOGY	Usually wide, bizarre and premature
PR INTERVAL	Buried in or following QRS complex	QRS INTERVAL	Greater than 0.12 seconds
P-P RATE	Underlying rhythm	R-R RATE	Underlying rhythm
RHYTHM	Irregular	RHYTHM	Irregular

Clinical Significance

A. Commonly occurs in
   a. Myocardial infarction
   b. Congestive heart failure
   c. Electrolyte imbalance
   d. Acid-base imbalance
   e. Drug toxicity
   f. Cardiac contusions

B. Frequent PVCs forewarn of, and possibly initiate, other more serious arrhythmia such as ventricular tachycardia and ventricular fibrillation

Treatment: Depends on the frequency, cause and patient toleration of the PVCs.

Goal: To decrease automaticity.

A. Treat underlying cause – correct electrolytes

B. Drug therapy:
   a. Amiodarone
   b. Lidocaine

IDIOVENTRICULAR RHYTHM (IVR)

General Information

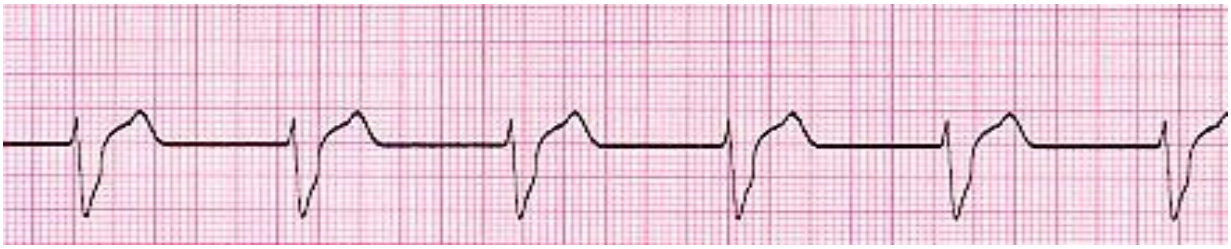
A. This is a general term which describes rhythms that are generated within the ventricular conduction system

B. The QRS complex is wide

C. Idioventricular rhythm is further described by the changes in ventricular rates

Idioventricular Rhythm			
Atrial Activity		Ventricular Activity	
P WAVE	Absent	QRS	Present
MORPHOLOGY	None	MORPHOLOGY	Usually wide, bizarre and premature
PR INTERVAL	None	QRS INTERVAL	Greater than 0.12 seconds
P-P RATE	None	R-R RATE	Underlying rhythm
RHYTHM	None	RHYTHM	Irregular

ACCELERATED IDIOVENTRICULAR RHYTHM (AIVR)



Accelerated IdioventricularRhtyhtm			
Atrial Activity		Ventricular Activity	
P WAVE	Absent	QRS	Present
MORPHOLOGY	None	MORPHOLOGY	Wide
PR INTERVAL	None	QRS INTERVAL	Greater than 0.12 seconds
P-P RATE	None	R-R RATE	40-100 bpm
RHYTHM	None	RHYTHM	Regular

Clinical Significance

A. Decreased cardiac output related to a slow ventricular rate may result in:
   a. Dizziness
   b. Fainting
   c. Weakness
   d. Hypotension

B. This rhythm can precipitate life-threatening tachyarrhythmias, such as ventricular tachycardia and ventricular fibrillation

Treatment: is always indicated

Goal: To maintain heart rate and cardiac output.

A. Drug therapy:
   a. Atropine
   b. Epinephrine Infusion
   c. Dopamine Infusion

B. Cardiac pacing

C. If no pulse: CPR, Epinephrine,Oxygen

VENTRICULAR TACHYCARDIA (V TACH or VT)

General Information

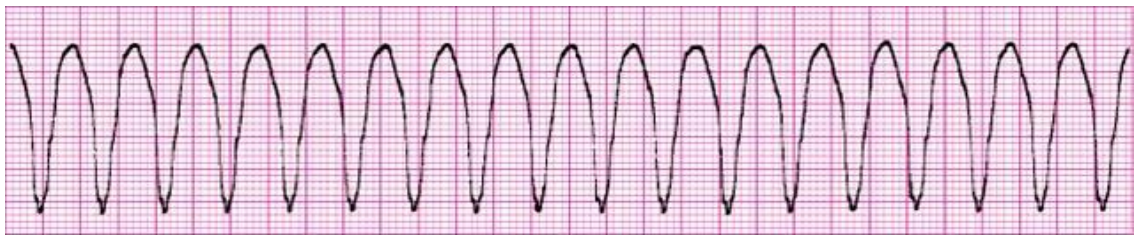
A. Three (3) or more consecutive PVCs is called ventricular tachycardia

B. The ventricular rate is greater than 100 bpm

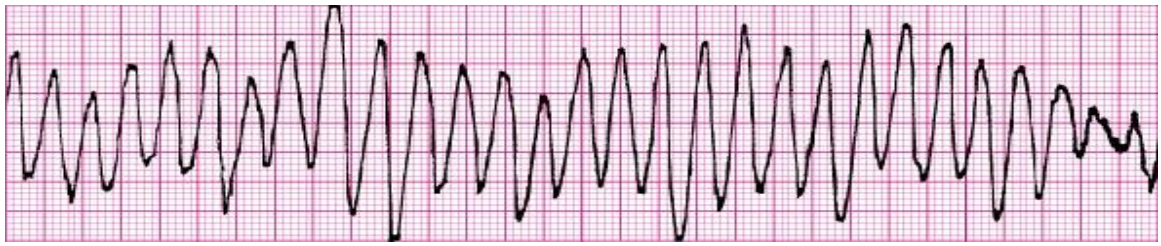
C. All irritable foci originate in the ventricles

D. Ventricular tachycardia is a life-threatening arrhythmia that requires *immediate* attention. A person in ventricular tachycardia may or may not have a pulse. This arrhythmia usually deteriorates to ventricular fibrillation

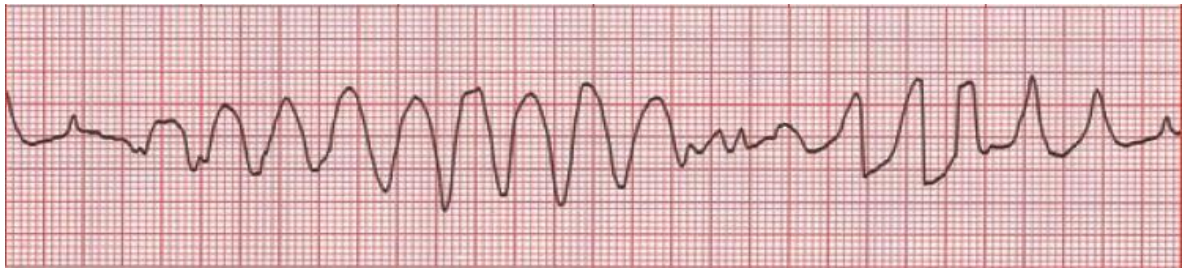
Monomorphic VT



Poly Morphic VT



Torsadepoint



Ventricular Tachycardia			
Atrial Activity		Ventricular Activity	
P WAVE	Present	QRS	Present
MORPHOLOGY	Not seen	MORPHOLOGY	Wide
PR INTERVAL	None	QRS INTERVAL	Greater than 0.12 seconds
P-P RATE	Unknown	R-R RATE	Greater than 100 bpm
RHYTHM	Unknown	RHYTHM	Regular/Irregular

Clinical Significance

A. Common in:
   a. Acute myocardial infarction
   b. Electrolyte imbalance
   c. Acid-base imbalance
   d. Drug toxicity
   e. Mechanical irritations of the heart:
      - Pulmonary artery catheter
      - Intra-aortic balloon pump

B. Usually results in a significant decrease in cardiac output

C. May progress to ventricular fibrillation and sudden death

Treatment: is always indicated.

Goal: To decrease automaticity of irritable ventricular foci, and improve cardiac output.

A. With pulse
   a. Stable: Amiodarone
   b. If Torsades de Pointes – Magnesium replacement
   c. Unstable: Synchronized Cardioversion
B. Without pulse (Pulseless VT)
   a. CPR
   b. Defibrillation
   c. Epinephrine
   d. Oxygen
   e. Anti-arrhythmic i.e. Amiodarone, Lidocaine

VENTRICULAR FIBRILLATION (V FIB or VF)

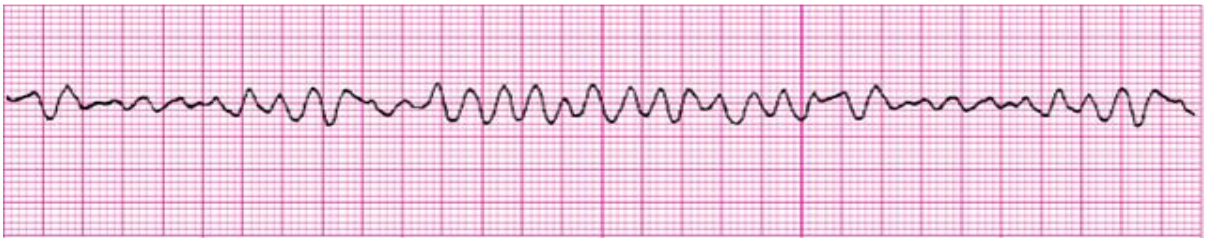
General Information

A. The ventricles are quivering, resulting in a disorganized, chaotic ventricular rhythm

B. The baseline is wavy and undulating

C. Coarse ventricular fibrillation is the initial rhythm. This deteriorates into fine ventricular fibrillation

D. This is a life-threatening arrhythmia



Ventricular Fibrillation			
Atrial Activity		Ventricular Activity	
P WAVE	Absent	QRS	Absent

MORPHOLOGY	None	MORPHOLOGY	Spiky/fine waves
PR INTERVAL	None	QRS INTERVAL	None
P-P RATE	None	R-R RATE	None
RHYTHM	None	RHYTHM	None

Clinical Significance

A. There is no pulse, no blood pressure and no cardiac output

B. Common in myocardial infarction, and open heart surgery

C. Results in sudden death

Treatment: is always indicated.

Goal: To restore organized electrical activity.

A. Immediate CPR and defibrillation.
B. CPR until defibrillation is available

a. Defibrillation
b. Epinephrine
c. Oxygen
d. Anti-arrhythmic i.e. Amiodarone

ASYSTOLE(AGONAL RHYTHM or DYING HEART)

General Information

A.Absence of all electrical and mechanical activity in the atria and the ventricles

A ystole

Atrial Activity		Ventricular Activity	
P WAVE	Absent	QRS	Absent
MORPHOLOGY	None	MORPHOLOGY	Wavy baseline
PR INTERVAL	None	QRS INTERVAL	None
P-P RATE	None	R-R RATE	None
RHYTHM	None	RHYTHM	None

Clinical Significance

A. No cardiac output
   a. No blood pressure
   b. No pulse

B. Common in myocardial infarction and drug overdose

C. Always results in death

Treatment: Always indicated.

Goal: To establish heart rate and cardiac output.

A. CPR

B. Drug therapy:
   a. Epinephrine
   b. Vasopressin

CARDIAC RHYTHM SUMMARY

Name	Rhythm	Rate(R-R)	P-Wave	QRS	PR Interval
Sinus Rhythm	Regular	60-100	Upright	0.06-0.12	0.12-0.20
Sinus Bradycardia	Regular	‹ 60	Upright	0.06-0.12	0.12-0.20
Sinus Tachycardia	Regular	100-160	Upright	0.06-0.12	
PSVT	Regular	150-220	May be flattened or notched. Seldom identifiable at rates above 200.May be lost in preceding T waves	0.06-0.12	May be smaller than 0.12 if P wave seen
Atrial Flutter	Regular (maybe irregular at times)	Atrial:250-400.Ventricular varies with Atrial conduction	Flutter waves: Saw tooth appearance	0.06-0.12	Not measurable
Atrial Fibrillation	Irregular	Slow Ventricular Response: Atrial 400600 Ventricular40-60 Controlled Ventricular Response:60-100 Rapid Ventricular Response:100-150	None	0.06-0.12	Not measurable
Sinus Arrhythmia	Irregular	60-100	Upright	0.06-0.12	0.12-0.20
Sinus Arrest/ Sinus Exit Block	Regular, but irregular where arrest occurs	60-100	Upright in underlying rhythm	0.06-0.12	Usually normal in underlying rhythm

Wandering Atrial Pacemaker (WAP)	Usually irregular	60-100, may be slower	Different directions, shapes and sizes	0.06-0.12	Varies due to shifting pacemaker
Premature Atrial Contraction (PAC)	Irregular	Is that of the underlying rhythm	Differs from the underlying rhythm	0.06-0.12,but can differ	May be normal but can differ to that of underlying rhythm
Junctional Rhythm	Regular	JR:40-60 Accelerated JR:60-100 Junctional Tachycardia:100-150	May occur before, during or after ORS complex. Always inverted if seen	0.06-0.12	If P wave present: usually less than 0.12
Name	**Rhythm**	**Rate(R-R)**	**P-Wave**	**QRS**	**PR Interval**
Premature Junctional Contraction (PJC)	Regular/Irregular, depending on underlying rhythm	Is that of the underlying rhythm	Differs from underlying rhythm, if seeninverted	0.06-0.12,but can differ	Differs from underlying rhythm
1st Degree AV Block	Regular	60-100	Upright	0.06-0.12	Greater than 0.20 but constant in every beat
2nd Degree AV Block-Type1	Irregular	Atrial rate greater than Ventricular	Normal but some P waves not followed by QRS	0.06-0.12	Gets progressively longer until QRS is dropped
2nd Degree AV Block-Type 2	Irregular	Atrial rate greater than Ventricular	Normal but some P waves not followed by QRS	0.06-0.12	May be normal or prolonged but is constant for each conducted beat
3rd Degree AV Block-Complete	Regular	Atrial greater than Ventricular	More P's than QRS's	0.06-0.12 junction wide if from ventricles	Atria and ventricles beat independent of each other. Maybe normal or prolonged but never constant in the

					conducted beats.
Idioventricular Rhythm (IVR)	Essentially Regular	IVR:20-40 Accelerated: above 40	Absent	Greater than 0.12	Not measurable
Premature Ventricular Contractions (PVC's)	Depends on underlying rhythm	Depends on underlying rhythm	Depends on underlying rhythm	Wide and bizarre	Depends on underlying rhythm
Ventricular Tachycardia	Usually regular	100-220	Absent	Wide	Not measurable
Ventricular Fibrillation	Chaotic	None	Absent	Absent	Absent
Ventricular Standstill	Atrial-regular Ventricular-none	Atrial:20-30	Present	Absent	None
Asystole	None	None	None	None	None

GLOSSARY AND ABBREVIATIONS

TERM	DEFINITION
Aberrancy	The flow of electrical current outside the normal electrical conduction pathways
Adrenergic	Refers to a division of the autonomic nervous system where norepinephrine is the neurotransmitter
AICD	Automatic implantable cardioverter defibrillators
Anticholinergic	An agent that opposes the effects of impulses conveyed by the parasympathetic nerves
Atrial kick	The amount of blood which is actively moved into the ventricles as a result of atrial contraction; accounts for 15-30% of the stroke volume
Arrhythmia	Literally absence of normal rhythm; often used interchangeable with the term 'dysrhythmia' to reflect any cardiac rhythm other than a sinus rhythm.
Autonomic nervous system	Also called the involuntary nervous system. Major functions include modulation of heart rate, control of blood pressure, intestinal motility, bladder function, and release of glandular secretions
AV dissociation	Atrioventricular dissociation is a general term that may be used to describe any cardiac dysrhythmia in which the atria and the ventricles beat independently of each other
Bpm	Beats per minute
Cardiac cycle	The time/distance between two complete QRS complexes
Cardiac output	The volume of blood pumped by the heart each minute. The principle factor in assessing cardiac performance
Catecholamine	A compound which mimics the actions of the sympathetic nervous system
Cholinergic	Refers to a division of the autonomic nervous system where acetylcholine is the principle neurotransmitter
Depolarization	Describes the movement of ions across the cell membrane, resulting in a predominantly positive field inside and a predominantly negative field on the outside of the cardiac cell
Ectopy	Arising from an abnormal site or tissue. Arrhythmias are considered ectopic when they originate outside the normal conduction pathways
EMD	Electromechanical dissociation, a form of pulseless electrical activity (PEA), occurs when the electrical conduction tissues generates an electrical pattern on the oscilloscope, but this is not transmitted into mechanical activity of the myocardium
Fib	Fibrillation
Foci	A point of electrical activity in the cardiac tissue other than the normal conduction system

Idioventricular	The origin of the impulse is below the AV junction and in the
TERM	**DEFINITION**
	ventricles. Idioventricular complexes will be wide
Inotropic	Refers to the force of cardiac contraction
LA	Left atrium
LV	Left ventricle
Ml	Myocardial infarction, 'heart attack'
Non-conducted p wave	Any time a p wave is not conducted through the node to the ventricles, it is termed non-conducted. Atrial activity is occurring without corresponding ventricular activity
Overdrive pacing	A form of artificial cardiac pacing used to override a fast ectopic electrical focus in the heart. Its intent is to inhibit that irritable focus, resulting in a slower heart rate.
Parasympathetic nervous system	One of two major divisions of the autonomic nervous system
PEA	Pulseless electrical activity
RA	Right atrium
Repolarization	Describes the electrical charge of the cardiac cell, when it is predominantly negative on the inside and positive on the outside; it is also known as the 'resting state'
Rpm	Respiration per minute
RV	Right ventricle
Stroke volume	The average volume of blood ejected from the heart with each contraction
Supraventricular	The origin of the impulses is above the ventricles. They are usually transmitted through the normal conduction pathways to the ventricles, and will usually produce a narrow, unchanging QRS complex

References

- Aehlert, B. (1995). *ECGs Made Easy*. United State of America: Mosby – Year Book.

- Marry-Evans, A. (2006). *ECG's Simply: Cardiac Arrhythmias Made Easy.*(1st ed). Gold Coast, Australia: Health Ed Professionals.

- Stock Toons, "n.d". Clipart. Retrieved December 11, 2008, from http://www.stocktoons.com/gallery/clipart/love_heart.html

- Skillstat, "n.d". Skillstat's learning: Tools. Retrieved November 20, 2008, fromhttp://www.skillstat.com/ECGskills.htm

- Urden, L.D. Stacy, K.M. and Lough, M.E. (2006).*Thelan's Critical Care Nursing: Diagnosis and management.*(5 ed). Philadelphia: ELSEVIER.