Basic
ECG
Rhythm
Interpretation

Textbook for OSU Medical Center
Basic ECG Dysrhythmia Course

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The Ohio State University Medical Center

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The Department of Educational Development and Resources at the Ohio State University Medical Center would like to acknowledge Sandra Walden MS, RN for her original creation of the Basic ECG Course and manual (1988; Revised 2002).

This manual is intended to be used as a reference only—it does not supersede OSUMC policy or physician orders.

**Applicable Policies:**

1. Pain and Symptom Management
2. ECG Monitoring, Continuous
3. Defibrillation, AED and Emergency Response
4. Pacemakers
5. Telemetry Beds, Adult Divisional Standards of Practice
Basic ECG Rhythm Interpretation

Objectives

At the completion of this course the learner will be able to:

1. Identify the sequence of normal electrical activation of the heart.

2. Describe the physiology of cardiac muscle contraction.


4. Identify the appropriate nursing and medical interventions, and first line medications for ECG rhythms.

5. Score at least 85% competency on the posttest.
GLOSSARY

**Aberrant Conduction** - Term applied to abnormal (slowed) intraventricular conduction of supraventricular (above the ventricles) impulses caused by a transient functional delay or block (slowed conduction) in one or more of the conducting fascicles (conduction system) causing a wide QRS.

**Arrhythmia** - Absence of heart rhythm

**Artifact** - Man-made disturbance in the EKG tracing; due to poor electrode contact or muscle activity originating outside the heart.

**Dysrhythmia** - A disturbance in the heart rhythm (abnormality)

**Ectopic Focus** - A site that initiates an electrical impulse other than the SA Node (usually prematurely due to irritability of the part of the myocardium)

**Escape Pacemaker** - (Backup) An alternate site capable of initiating an electrical impulse to ultimately stimulate a heart beat when the SA Node fails to do so.

**Electrodes** - Sensing devices placed on the chest (or extremities) for the purpose of detecting electrical impulses traveling through the heart.

**Focus** - Site of impulse formation

**Infranodal** - Adjective referring to focus below the AV Node.

**Isoelectric Line** - The imaginary line on which the ECG waves are drawn; also called the baseline or PR Line.

**Multifocal** - Adjective describing ectopic foci that originates from multiple sites, thereby causing configurations (shapes) different from each other.

**Multiformed** – Adjective describing multiple morphology (shape) of the qRs in a rhythm. The shapes vary therefore the likelihood is increased that the ectopic foci originate from multiple sites. (Multiformed is preferred over multifocal).

**Retrograde Conduction** - An electrical impulse travels the opposite direction from normal. It is usually associated with an ectopic focus that occurs in the Junction, forming a retrograde P wave. The P wave will be inverted and either is seen before or after the QRS or is not seen at all.

**Supraventricular** - Adjective referring to a dysrhythmia originating above the ventricles (SA Node or Atria), usually characterized by a qRs complex of normal width.

**Unifocal** - Adjective describing an ectopic focus that originates from one focus. The complexes seen are uniform.

**Uniform** – Adjective describing the uniform shape of the qRs complexes in a rhythm

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**Frequently Used Abbreviations**

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Chapter One

Basic Principles

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Chapter One

BASIC PRINCIPLES

OBJECTIVES

1. Review anatomy of the heart.

2. Describe two myocardial cell types.

3. Describe four properties of cardiac cells.

4. Discuss the process of depolarization and repolarization.

5. Describe the sequence of electrical activation of the heart.

6. Correctly label the ECG complex.

7. Demonstrate computation of the heart rate on a rhythm strip, using two different methods.

8. Identify four potential pacemakers in the heart.

9. Analyze a rhythm strip in a systematic fashion.
CARDIAC ANATOMY:

The heart is a muscular pump. The left side of the heart is the larger and thicker side. It does more work because it pumps oxygenated blood into the aorta and then throughout the entire body. The thinner, right side of the heart pumps the same amount of blood, but only has to send it a short distance into the lungs via the pulmonary arteries. Each side of the heart consists of two chambers, an atrium and a ventricle. The thick walled ventricles are the larger pumping chambers that expel blood from the heart with each beat (contraction or systole.) The relatively thin-walled atria function as collecting and loading chambers. The atria hold blood being returned to the heart during ventricular systole. In between contractions, the ventricles are relaxed (diastole). During diastole, blood flows into the ventricles from the atria, at first passively, and then propelled by atrial contraction (atrial systole). Strategically located valves prevent backflow of blood. The mitral valve is between the left atrium and ventricle, the aortic valve between the aorta and left ventricle, the tricuspid valve between the right ventricle and right atrium and the pulmonary valve between the main pulmonary artery and the right ventricle.

The endocardium is a layer of smooth lining cells. These cells are found not only in the heart but also on the inside of all the blood vessels of the body, where they are called endothelium. The myocardium is the mass of heart muscle cells whose coordinated contraction causes the chambers of the heart to contract and pump blood. The myocardium is thin in the atria, thicker in the right ventricle and thickest in the left ventricle. The epicardium is a fatty layer on the outer surface of the myocardium. The major coronary blood vessels, the vessels that supply blood to the heart itself, run through the epicardium. The outermost layer is the pericardium, actually two layers with a small amount of lubricating fluid between them, forming the pericardial sac, which encloses the entire heart.
CORONARY CIRCULATION

The heart is always in continuous action. In order to maintain aerobic metabolism and contractile activity, the heart must be able to receive oxygen and nutrients and have carbon dioxide and other wastes removed. This is accomplished through coronary circulation. The heart is supplied by 2 major coronary arteries: the right coronary artery (RCA) and the left coronary artery (LCA). These arteries arise from the aorta immediately distal to the aortic valve. Coronary perfusion occurs during diastole. The RCA supplies the right atrium, right ventricle, and a portion of the posterior and inferior surfaces of the left ventricle. The RCA supplies the AV node and bundle of His in 90% of hearts and the sinus node in 55% of hearts. The LCA is the main coronary artery that passes between the left atrial appendage and the pulmonary artery and then divides into two major branches, the left anterior descending artery (LAD) and the left circumflex artery (LCA). The LAD is responsible for supplying portions of the left and right ventricular myocardium and the majority of the interventricular septum. The (LCA) supplies the lateral wall and posterior wall of the left ventricle, SA node (45%), AV node (10%), and posterior inferior portion of the LBB.

With knowledge of coronary anatomy, we can anticipate complications of various coronary artery blockages. Since the RCA supplies the SA Node in 55% of hearts and the AV Node in 90% of hearts, we can anticipate problems that would arise with a compromise of these primary pacemakers such as sinus bradycardia, sinus arrest, junctional rhythms and AV Block rhythms. Since the LCA perfuses the anterior wall of the heart, we can anticipate problems that would result from a lack of pump function, such as congestive heart failure, cardiogenic shock, and ventricular irritability. It is important to understand which areas of the heart are perfused by which coronary artery, and to anticipate the possible complications of an occlusion.

CARDIAC CYCLE

Blood is propelled to the body by the sequential activation of the cardiac chambers through electrical and mechanical factors. The cardiac cycle is divided into two major phases, ventricular systole and diastole.

Ventricular Systole:
Ventricular contraction follows the ventricular depolarization reflected by the electrocardiographic QRS wave. Ventricular pressure increases rapidly. This results in the opening of the pulmonic and aortic valves. At the onset of this period, pressures in the atrium and ventricle are approximately equal, but atrial pressure decreases with atrial muscle relaxation and repolarization. The tricuspid and mitral valves are closed as the atria fill with blood.

Ventricular Diastole:
Ventricular muscle relaxation begins, pressure decreases, ventricular filling occurs as the tricuspid and mitral valves open and blood flows from the atria into the relaxed ventricles. During diastole, approximately 75% of blood flows passively from the atria through the open tricuspid and mitral valves into the ventricles even before the atria contract. Atrial contraction (atrial kick) contributes an additional 25% to ventricular filling.
CARDIC OUTPUT

Cardiac output is the amount of blood the left ventricle pumps into the aorta per minute. This is a systolic event. The stroke volume times the number of cardiac cycles per minute (heart rate) equals the cardiac output. The ejection fraction (EF) is the percentage of total ventricular volume injected during each contraction. Normally it is greater than 55% and usually is approximately 65%. Three factors affect or determine stroke volume:

**Preload**: is the degree of stretch, or tension, on the muscle fibers when they begin to contract.

**Afterload**: is the load or amount of pressure the left ventricle must work against to eject blood during systole and corresponds to the systolic pressure. The greater the resistance is, the greater the heart’s workload. Afterload is sometimes called systemic vascular resistance.

**Contractility**: is the ventricles’ ability to contract, which is determined by the degree of muscle fiber stretch at the end of diastole. The more the muscle fibers stretch during ventricular filling, up to an optimal length, the more forceful the contraction.

Frank-Starling law of the heart

This law defines the fundamental principle of cardiac behavior which states that the force of contraction of the cardiac muscle is proportional to its initial length. The energy set free at each contraction is a simple function of cardiac filling. When the diastolic filling of the heart is increased or decreased with a given volume, the displacement of the heart increases or decreases with this volume.

**BASIC ANATOMY OF THE CONDUCTION SYSTEM**

Stimulation of the heart originates in the Sympathetic and Parasympathetic branches of the Autonomic Nervous System.

The impulse travels first to the Sinoatrial Node (SA Node) located in the posterior wall of the right atrium. The SA Node is the main cardiac pacemaker from which wave-like impulses are sent through the atria stimulating first the Right and then the Left Atrium. The SA Node is richly supplied with both Vagal and Sympathetic nerve fibers and the former are derived mainly from the right Vagus Nerve.

Under normal circumstances the **SA Node will initiate electrical impulses at the rate of 60 to 100 times per min.**

Physiologic and Anatomic studies indicate that the Sinus impulse spreads in the Atria and to the AV Node through the three (3) Atrial internodal tracts.
1. The anterior internodal tract divides into two parts, one going to the left atrium and known as Bachman's Bundle, the other descending to the upper margin of the A-V node.

2. The middle internodal tract, or Wenckebach Bundle, which descends within the interatrial septum to enter the upper part of the A-V node.

3. The Posterior Internodal Tract--or Thorel's tract reaches the interatrial septum and enters the posterior margin of the AV node.

As soon as the Atria have been stimulated, the impulse slows as it passes through the Atrioventricular (AV) Node, which lies in the inferior posterior portion of the Right Atrium. The AV node has two vital functions in serving the heart.

1. The AV Node has the ability to protect the ventricles from excessively fast rates that may originate in the atria.
2. The area just above and just below the AV Node (AV Junction) like the SA node, may also serve as the pacemaker for the heart, should the SA node or atrial tissue fail to initiate impulses or should the impulse fail to reach the AV node.

The AV Junction will normally initiate impulses at 40 to 60 times per minute when functioning as the pacemaker. (AV Junction includes the A-N region, the AV Node and the N-H region- see AV Junction figure on Page 11). The AV node itself is totally devoid of pacemaking cells.

The wave of excitation then spreads to the Bundle of HIS (common bundle) which is continuous with the AV node and reaches the interventricular septum where it divides into the right and left bundle branches.

The right bundle branch runs along the right side of the interventricular septum, and reaches the base of the anterior papillary muscle of the right ventricle where it divides into a network supplying the right ventricular myocardium.

The left bundle branch runs along the left side of the interventricular septum and divides almost immediately into an anterior (superior) division and an inferior (posterior) division. The anterior division, a relatively long and thin pathway reaches the base of the anterior papillary muscle and supplies the anterior superior part of the left ventricle. The posterior division, a relatively short and thick structure, passes to the base of the posterior papillary muscle and supplies the inferior posterior aspect of the left ventricle. The intraventricular conduction system is therefore composed of three conduction pathways, also called fascicles; the right bundle branch, the anterior division of the left bundle branch, and the posterior division of the left bundle branch.

The right bundle branch and both the anterior and posterior divisions of the left bundle branch divide into a complex network of fibers, called the Purkinje fibers, which is distributed to the ventricular myocardium. The fibers are more abundant in the subendocardial layers and are longer than the common myocardial fibers. The Purkinje fibers in the human heart are believed to lack nerve supply entirely.
The ventricles also have the ability to function as a backup pacemaker (escape mechanism), should they not receive impulses from the S-A node, atrial tissue or AV node. The ventricles may be considered a much less efficient backup system, for the rate at which they normally initiate impulses (inherent rate) is 20-40 times per minute.

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ELECTROPHYSIOLOGICAL PROPERTIES

PROPERTIES of CARDIAC CELLS
Cardiac cells have three electrical properties:
1. Automaticity
2. Excitability
3. Conductivity

AUTOMATICITY: The heart has the property of initiating and maintaining rhythmic activity without the help of its neurological supply. The myocardial cells that possess the property of automaticity are called pacemaking cells.

Under normal circumstances the pacemaking cells with the highest degree of automaticity and more rapid discharge rate are located in the Sinus Node, the primary pacemaking focus of the heart. Pacemaking cells with a lesser degree of Automaticity and slower inherent or natural discharge rate are present in the atria, in the AV Junction and in the ventricles; forming auxiliary ectopic pacemaking foci (sites). The more peripheral the site of the pacemaking focus from the SA Node, the slower its automaticity and inherent discharge rate.

EXCITABILITY: Both pacemaking and non-pacemaking myocardial cells have the property of responding to a natural or artificial electrical stimulus. This ability to respond to an impulse or stimulus is called excitability.

CONDUCTIVITY: Conductivity is the property that allows the myocardial cell to propagate an impulse to a neighboring cell. An impulse of adequate strength originating in any area of the heart during its resting period creates a wave of excitation that is propagated over the whole tissue.

The cardiac cells also have one mechanical property:
1. Contractility

CONTRACTILITY: Contractility is the ability of the cardiac muscle to respond to an electrical impulse with pump action. (the ability of the muscle to shorten and contract).

DYSRHYTHMIAS
The terms dysrhythmia and arrhythmia are used interchangeably in the literature. Since dysrhythmia means an abnormality in rhythm, whereas arrhythmia indicates an absence of rhythm, the former term will be used in this text.

Cardiac dysrhythmias may result from one of the following mechanisms:
- Disturbances in automaticity
- Disturbances in conductivity
- Combinations of altered automaticity and conductivity.
ELECTRICAL ACTIVITY OF THE HEART

In order to better understand disturbances in the electrical activity of the heart, it is important to have a basic knowledge of the electrical properties of the working and electrical system cells.

MYOCARDIAL CELL TYPES: There are two basic groups of cells within the myocardium which are important for cardiac function:

1. Working Myocardial Cells - possess the property of contractility, or the ability to shorten and then return to their original length.

2. Electrical System Cells - Cells belonging to the electrical system that are responsible for conduction of the electrical stimulus to the working cells of the myocardium.

Working Cells (Contraction of the Myocardial Cells) - Cardiac muscle cells are polarized during the resting state. During resting the cells have a negative charge inside the cell membrane and a positive charge outside the cell membrane. When the cell membrane is electrically stimulated a process called depolarization occurs. Electrical stimulation of the cell results in a change in the electrical properties of the cell (depolarization) by causing the flux of positive ions into the cell and negative ions outside the cell membrane. Once depolarized, the cell shortens and contracts. Next the cell repolarizes returning the negative ions inside the cell membrane and positive ions outside the cell. Once repolarized, the cell returns to the resting state ready for depolarization and repolarization to occur again.
**Electrical System Cells** – The electrical wave is propagated from cardiac cell to cell and fiber to fiber through the heart via the electrical system. The rate at which the impulse travels (conduction velocity) varies. The conduction velocity is the slowest through the AV Node; Ventricular muscle conducts 6 times the speed of the AV Node; Atrial muscle at 20 times the speed of the AV Node velocity; and the Bundle Branches and Purkinje Fibers conduct at 40 times the rate of the AV Node. This rapid conduction through the entire electrical system occurs normally and contributes toward an organized and efficient contraction.

Should a site outside the electrical system initiate an impulse (for example, a ventricular site), a different mechanism of depolarization occurs. Because the impulse occurs outside the specialized electrical system cells, asynchronous activation of the right and left ventricle occurs and in addition the conduction velocity is slowed through the myocardial cells. This results in a wide bizarre qRs.

**THE ELECTROCARDIOGRAM**

An electrocardiogram is a graphic tracing of voltage produced by depolarization and repolarization of the heart muscle. It is important to remember that the ECG shows you only the electrical activity of the heart – it shows nothing related to the mechanical function of the heart muscle.

When no current is flowing, the stylus (writing point) of the electrocardiograph does not move, and a straight line is recorded. A current flow causes a deflection (movement) of the stylus, so that a wave is inscribed on the ECG paper. Normal movements from the baseline are called waves and are designated P, q, R, s, T and U. The spaces between the waves, which usually appear as straight lines, are called segments and are named by the waves they separate (for example, ST segment between the S and T waves). Intervals are also named by the waves at their beginning and end, for example, PR interval, but their measurement is from the beginning of the first wave, unlike the segments whose measurement starts at the end of the first wave. An interval ends with the start of the wave at its end just like a segment does, with one exception; the qT interval is measured from the start of the q wave to the end of the T wave. A complex includes more than one wave (qRs complex).

Each wave recorded on the electrocardiogram corresponds to a particular event in the cardiac cycle. Although the heart consists of several layers, it is only the heart muscle cell layer (myocardium) that generates currents large enough to be recorded by the electrocardiograph.

The current recorded by the electrocardiograph at any moment is the sum of all the currents flowing in cells throughout the heart at the particular instant.

Deflections that emanate above the isoelectric line are called positive deflections. Deflections that radiate below the isoelectric line are considered negative deflections.
ELECTRODE PLACEMENT

Electrodes are small patches which are placed on the chest to transmit electrical activity from the heart to the telemetry system. Correct electrode placement is critical in order to correctly identify cardiac rhythms and rhythm disturbances. “Research has shown that when an electrode is misplaced by 1 intercostal space, the morphology of the qRs can change dramatically and missed …diagnosis may occur… There are certain leads that are appropriate for specific dysrhythmias. For example, leads V1 and V6 are the leads of choice to differentiate Ventricular Tachycardia from SVT” (AACN Practice Alert: Dysrhythmia Monitoring, 2004). Telemetry devices can be portable and fit into the pocket of a gown, or they can be hardwired to the central nursing station or a bedside monitor. There are 3, 5 and even 12-lead bedside systems. If you have the capability with your monitoring system, it is always wise to display two leads from two different areas of the heart to maximize your ability to capture ectopy or ECG changes.

Skin preparation is another important factor in the quality of an ECG. The skin should be cleaned with soap and water or alcohol and allowed to dry. Gentle abrasion of the electrode sites with a washcloth aids with impulse transmission. It may be necessary to shave the sites. Before applying the electrodes, ensure that the conductive gel is moist and intact, and attach the lead wires. Avoid bony prominences.
Normal ECG Complex

The wave of depolarization spreading through the heart can be recorded on paper. This is called the electrocardiogram ECG (EKG). The electrical stimulus originating from the SA Node proceeds away from the node concentrically in all directions in an enlarging circular wave. This electrical impulse spreads across the atria and yields a P Wave on the ECG. The P wave represents atrial depolarization electrically.

![P Waves diagram]

The impulse then reaches the AV Node, where conduction is the slowest. The impulse takes 1/10 second to travel through the node, allowing blood to enter the ventricles. This time frame is shown on the ECG as the P-R interval, which is measured from the onset of the P wave to the beginning of the QRS complex.

**The normal P-R interval is 0.12 - 0.20 seconds**
After the 1/10 second pause, the AV Node is stimulated, initiating an electrical impulse that starts down the AV bundle into the Bundle Branches, to the Purkinje fibers and into the myocardial cells, causing ventricular depolarization. The electrical activity of the stimulation of the ventricles is represented by the qRs complex. The q wave is the first downward stroke of the qRs complex and is followed by the upward R wave. The q wave is often not present. The upward R wave is followed by a downward s wave. This total complex duration represents the electrical activity of ventricular depolarization and contraction.

**Normal duration of the qRs is .04 – 0.10 seconds**

Variation of qRs Complexes

There is a pause after ventricular depolarization that is represented by the ST segment, which normally is on the isoelectric (flat) line having no voltage from the end of the S wave to the beginning of the T wave.
Recent studies have shown the importance of continuous ST segment monitoring, especially in patients with “acute coronary syndromes presenting to the ED, patients undergoing a catheter-based procedure, patients with a cardiac history undergoing a surgical procedure and patients in the ICU following cardiac surgery…” (AACN Practice Alert: ST Segment Monitoring, 2004). Continuous ST segment monitoring allows for “…continuous monitoring of ST segment for changes associated with ischemia to allow early indications of ischemia even in the absence of chest pain” (Dennison, 2000, p.56). It is important to realize that elevation or depression of just 1mm is considered significant. Just as with dysrhythmia detection, in which leads V1 and V6 were optimal, leads III and V3 are optimal to detect ST segment deviations. However, if the patient has had a 12-lead ECG, you can determine which lead will be the most appropriate based on their individual ischemic event. Typically, ST segment elevation represents injury or severe ischemia, and ST segment depression represents ischemia.

The repolarization of the ventricles then occurs so that heart cells can regain the negative charge and, thus, may depolarize again. Final repolarization is represented by the T wave.
The q-T interval is measured from the onset of the qRs complex to the completion of the T wave. The normal duration of the q-T (with a normal rate 60-100) is generally 0.35-0.45 seconds. The typical qT interval should not exceed more than one-half of the preceding R-R interval.

Count the number of small boxes between two consecutive R waves and divide by 2. Count the number of small boxes in the qT interval. If the qT is longer than ½ of the R-R measurement, the qT is prolonged. This method is called the qTc (or the qT calculated to take heart rate into account) and is the most accurate method of determining the length of the qT interval. The Corrected qT (qTc) equation is qT/sqrt (RR).

The qT interval should be calculated with each rhythm interpretation according to hospital or unit policy (see policy ECG Monitoring, Continuous), and be monitored for trends. A prolonged qT interval shows that there is a delay in ventricular repolarization. This means that the relative refractory period (the most vulnerable part of the cardiac cycle) is prolonged and this can place the patient at risk for Torsades de Pointe. Some references site a qTc of > 0.50 seconds as a risk for Torsades de Pointe. Specific risk factors include certain medications such as Amiodarone, hypokalemia, low magnesium and calcium levels, bradydysrhythmias, and hypothermia.
A U wave, a small wave of low voltage, is sometimes seen following the T wave. It is often best discerned in lead V3. It is rendered more prominent by potassium deficiency, and its polarity is often reversed in myocardial ischemia and left ventricular strain. The U Wave's precise significance is uncertain. In the cardiac cycle it coincides with the phase of supernormal excitability during ventricular recovery, and in this connection it is interesting to note that most ventricular premature beats occur at about the U wave. It is important to distinguish U waves from a second P wave.
P-QRS-T-U Waves Summary

P Waves
- Represent atrial depolarization
- Should be smooth and round
- Should be upright in all leads except aVR
- May be diphasic or biphasic (both positive and negative components)
- May be notched or peaked
- Should be one P for every qRs complex

PR Interval
- Normal duration = 0.12 - 0.20 seconds
- Measured from the beginning of the P to the beginning of the qRs
- Represents atrial depolarization + delay through the AV Node

qRs Complex
- Normal duration 0.10 seconds or less
- Measured from the beginning of the qRs complex to the end of the S (the J-point)
- Represents ventricular depolarization
- Atrial repolarization is hidden within the qRs
- q = first downward deflection before the R wave
- R = first initial upward deflection following the P wave
- s = first downward deflection following the R wave; the J-point is the point at which the s wave returns to baseline

ST Segment
- Period between the completion of ventricular depolarization and beginning of final ventricular repolarization.
- Normally found on the isoelectric line; may be elevated or depressed

T Wave
- Represents recovery phase
- Normally upright in all leads except aVR
- Round and asymmetrical

U Wave
- Occurs after the T wave (not always present)
- Important to distinguish from second P wave

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)
Each rhythm strip should be analyzed in a systematic fashion. It is helpful to consider the Five Steps of ECG Rhythm analysis.

The Five Steps of ECG Rhythm Analysis

1. Determine regularity
2. Calculate Heart Rate
3. Examine P-\textit{q}Rs-T waves
4. Measure Intervals
5. Identify the Rhythm

**RHYTHM**

The rhythm is said to be regular when there is a constant distance between similar waves (P-P or R-R). To determine whether the rhythm is regular or irregular place the straight edge of a piece of paper along the baseline of the rhythm strip. Then, move the paper up slightly so that the straight edge is near the top peak of the P waves. With your pencil, make a dot on the paper at each of two consecutive P waves; this is the P-P interval. Now, move the paper across the strip from left to right lining up the dots with each consecutive P wave. If each distance between all the P waves is the same, the atrial rhythm is regular, if the distance varies, the atrial rhythm is irregular. Next, using the same method, measure the distance between consecutive R waves (the R-R interval) to determine whether the ventricular rhythm is regular or irregular.
Calipers may also be used to achieve measurement (a more accurate technique).

The rhythm is considered regular if all the R-R measurements are consistent. If the rate is within a normal range of 60-100 beats per minute, and the R-R interval varies no more than 3 small boxes (0.12 sec), the rhythm is considered essentially regular (even though it is slightly irregular). This 0.12 second leeway in regularity does not apply to rates above 100.
ECG PAPER
The ECG is recorded on ruled paper. The smallest divisions are one millimeter squares (one mm long and one mm high). There are five small squares between the heavy black lines. The height and depth of a wave is measured in millimeters and represents a measure of voltage.

The horizontal axis represents time. The amount of time represented by the distance between the heavy black lines is 0.20 seconds. There are five small squares between the heavy black lines. Therefore each small square represents 0.04 seconds. By measuring along the horizontal axis the duration of any part of the cardiac cycle can be determined.

6 seconds = 30 Large or 150 Small Boxes
The rate of any cardiac rhythm can be quickly determined from an ECG strip. Once you understand the layout and measurement of the graph paper, rate calculations become easy. Most ECG recorders in North America run ECG paper at 25mm/sec. (Remember Large boxes = 0.20 seconds in time and small boxes = 0.04 seconds in time). Three methods for determining heart rates will be discussed. The method used for rate measurement is determined by whether the rhythm is regular or irregular. If the rhythm is regular the Grid or Ruler Method can determine the rate accurately. If the rhythm is irregular the best means for determining the rate is the Scan Method.

### Time Small boxes (1 = 0.04 seconds) Large Boxes (1 = 0.20 seconds)

<table>
<thead>
<tr>
<th>Time</th>
<th>Small boxes</th>
<th>Large Boxes</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.04 seconds</td>
<td>1 small box</td>
<td></td>
</tr>
<tr>
<td>0.20 seconds</td>
<td>5 small boxes</td>
<td>1 large box</td>
</tr>
<tr>
<td>1 second</td>
<td>25 small boxes</td>
<td>5 large boxes</td>
</tr>
<tr>
<td>3 seconds</td>
<td>75 small boxes</td>
<td>15 large boxes</td>
</tr>
<tr>
<td>6 seconds</td>
<td>150 small boxes</td>
<td>30 large boxes</td>
</tr>
<tr>
<td><strong>60 seconds</strong></td>
<td><strong>1500 small boxes</strong>*</td>
<td><strong>300 large boxes</strong>*</td>
</tr>
</tbody>
</table>

* = Number of small and large boxes in a minute

### 1. Grid Method
The rate can be determined by counting the number of boxes, small or large, between R waves and then dividing that number into 1500 or 300 respectively. (See example strips below)
1. **The Grid Method** is done by either dividing 1500 (1500 smaller, 1mm, squares in one minute) by the number of small boxes between 2 R waves or by dividing 300 (300 large, 5mm, squares in one minute) by the number of large boxes between 2 R waves. The Grid method is the most accurate, yet very time consuming to calculate and impractical when trying to get a quick estimate of the heart rate. The large box method can be completed quickly by memorization. The Grid Method is limited to regular rhythms.

<table>
<thead>
<tr>
<th>Large Squares Between R Waves</th>
<th>Rate Per Minute</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Large Square</td>
<td>300</td>
</tr>
<tr>
<td>2 Large Squares</td>
<td>150</td>
</tr>
<tr>
<td>3 Large Squares</td>
<td>100</td>
</tr>
<tr>
<td>4 Large Squares</td>
<td>75</td>
</tr>
<tr>
<td>5 Large Squares</td>
<td>60</td>
</tr>
<tr>
<td>6 Large Squares</td>
<td>50</td>
</tr>
<tr>
<td>7 Large Squares</td>
<td>42</td>
</tr>
</tbody>
</table>

2. **The Scan Method** is done by counting the number of R waves in a 6 second strip and then multiplying by 10 (6 seconds x 10 = 60 seconds; therefore the number of R waves per 6 second strip multiplied by 10 gives the rate/minute). The scan method is the most practical and commonly used because calculations can be determined for both regular and irregular rhythms quickly.

5 R waves/6 second strip = 5 x 10
Rate = 50/minute
3. Three Beat Ruler Method – is done by using a commercially developed ruler. Place the first qRs complex on the “0”. Next, count three complexes over; the rate on the ruler corresponds with the third complex.

![Rate = 50](image)

Step 3 Examine P-qRs-T Waves

**P WAVES**
After assessing the rate and rhythm, the next step is to examine the P waves. Determine the following:
- Are P waves present and in front of all qRs complexes?
- Does a P wave follow the qRs complex?
- Is there 1P:1qRs relationship?
- Are the P waves all a similar shape?
- Are the P waves all upright (positive) in all leads except aVR?

**qRs complexes**
Next determine the following:
- Are the qRs complexes present with each p wave?
Step 4 Measure PR Intervals and qRs Complexes

PR INTERVAL
The next step is to measure the PR Interval. The PR interval begins with the initiation of the P wave (where the slope of the P wave first leaves the isoelectric line) and ends with the first deflection of the qRs complex.

- Are PR Intervals within normal limits (0.12 seconds – 0.20 seconds)?
- Are PR Intervals consistent (constant)?

qRs Duration
The qRs should measure between 0.04 seconds up to 0.10 seconds (not equal to or greater than 0.12 seconds). The qRs begins at the point where the complex begins (either with a q or R) and ends with the J point (sometimes referred to as the J junction). The J point is defined as the point at which the s wave returns to baseline and the ST segment begins.

- Normal qRs duration is 0.04 seconds – 0.10 seconds

QT Intervals – The Q-T interval is measured from the first initiation of the qRs complex to the end of the T wave. Normal Q-T intervals are based on rate and gender. The Q-T grows shorter as the rate increases. (See chart on next page)
Normal Q-T Intervals

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Men &amp; Children</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>40</td>
<td>0.45-0.49 seconds</td>
<td>0.46-0.50 seconds</td>
</tr>
<tr>
<td>50</td>
<td>0.41-0.45 seconds</td>
<td>0.43-0.46 seconds</td>
</tr>
<tr>
<td>60</td>
<td>0.39-0.42 seconds</td>
<td>0.41-0.43 seconds</td>
</tr>
<tr>
<td>71</td>
<td>0.36-0.38 seconds</td>
<td>0.37-0.41 seconds</td>
</tr>
<tr>
<td>100</td>
<td>0.31-0.34 seconds</td>
<td>0.32-0.35 seconds</td>
</tr>
<tr>
<td>150</td>
<td>0.25-0.28 seconds</td>
<td>0.26-0.28 seconds</td>
</tr>
<tr>
<td>172</td>
<td>0.23-0.26 seconds</td>
<td>0.24-0.26 seconds</td>
</tr>
</tbody>
</table>

Step 5 Identify the Rhythm

Once all of the steps have completed and the information examined, a correct and complete interpretation can be made. The steps are summarized below, along with normal ranges noted.

- **Rate** = 60 – 100 per minute
- **Rhythm** = Regular
- **P waves** = Present, similar, 1 and only 1 P for each qRs
- **PRI** = 0.12 – 0.20 seconds and consistent
- **qRs** = 0.04 – 0.10 seconds
- **P: qRs** = One P for each qRs
Chapter Two

Rhythms Originating from the Sinus Node

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Objectives Chapter Two

1. Identify normal Sinus Rhythm.

2. Compare and contrast Normal Sinus Rhythm, Sinus Bradycardia and Sinus Tachycardia.

3. Differentiate normal Sinus Rhythm from Sinus Arrhythmia and Sinus Pause or Sinus Arrest.
The Sinus Complex

The salient point for identifying a complex from the Sinus Node is the presence of a **normal P wave**. In lead II normal P waves will have a shape that is rounded, upright and similar to each other. A normal P wave in Lead aVr will be inverted; and in V1 may be biphasic (have both a negative and positive component).

![Normal P Wave Lead II](image1)

![Normal P Wave Lead aVr](image2)

![Normal P Wave Lead V1](image3)

The normal qRs will also vary from lead to lead. It should be upright or positive in Lead II. Most normal qRs complexes in Lead II will not have a visible q Wave, while a normal q wave may be present in Leads I, aVI, V5 & V6.

![Normal qRs Lead I](image4)

Therefore any complex that has originated from the SA Node will have this normal P-qRs-T configuration.
If the impulse from the SA Node is normally conducted down through the atrial tracts, the AV Node, the Bundle of HIS, the bundle branches and the purkinje system, the sinus complex will have normal PR interval (0.12-0.20 second) and qRs duration (0.04-0.10 second). Every complex arising from the SA node that is conducted normally will have similar morphology.

Sinus rhythms have the following ECG characteristics in common: One Sinus P wave for each qRs and similar to each other, PR and qRs intervals normal and constant PR intervals. ECG characteristics that vary are rate and rhythm. Sinus rhythms include:

- Normal Sinus Rhythm
- Sinus Tachycardia
- Sinus Bradycardia
- Sinus Dysrhythmia
- Sinus Pause (Arrest)

**Sinus Rhythms ECG Summary**

- **Rate:** 60-100, >100 or < 60 per minute
- **Rhythm:** R- R = or ≠
- **P Waves:** Upright (except AVR); similar
- **P-R interval:** 0.12 -0 .20 seconds & consistent
- **qRs:** 0.04 – 0.10 seconds
- **P to qRs:** 1P:1qRs
Normal Sinus Rhythm (NSR)

Normal sinus rhythm represents a regular discharge of the sinus node with subsequent atrial depolarization at a rate between 60-100 per minute. The rhythm is regular (or slightly irregular-no more than 3 small boxes or 0.12 seconds difference in R-R intervals when the rate is 60-100).

**ECG Summary**

- **Rate:** 60-100 per minute
- **Rhythm:** R- R = (no more than 0.12 seconds difference in R-R when the rate is 60-100)
- **P Waves:** Upright (except AVR); similar
- **P-R interval:** 0.12 -0 .20 seconds & consistent
- **qRs:** 0.04 – 0.10 seconds
- **P to qRs:** 1P:1qRs

**Normal Sinus Rhythm**

- Rate: 80
- Rhythm: R – R = PRI: 0.16 - 0.18 seconds
- qRs: 0.08 seconds

= P Waves
= R Waves
= T Waves
Sinus Tachycardia

Sinus Tachycardia represents an increase in the rate of discharge of the sinus node. It may be secondary to multiple factors and is a physiologic response to a demand for a higher cardiac output.

**ECG Summary**

- **Rate:** > 100 (usually not > 150)
- **Rhythm:** R- R =
- **P Waves:** Upright (except AVR); similar
- **P-R interval:** 0.12 -0.20 seconds & consistent
- **qRs:** 0.04 – 0.10 seconds
- **P to qRs:** 1P:1qRs

**Causes of Sinus Tachycardia:**

- Exercise
- Fever
- Anxiety, Fear
- Fight or Flight
- Hypovolemia
- Hypoxia
- Acute MI
- Congestive Heart Failure
- Medications
- Substances

**Therapy:**

- None specific for this dysrhythmia
- Assess patient to determine stability and notify the physician if new rhythm and/or unstable
- Treat underlying cause
Sinus Bradycardia

Sinus Bradycardia is a decrease in the rate of atrial depolarization subsequent to a slowing of the sinus node. It may be secondary to intrinsic sinus node disease, increased parasympathetic tone, or drug effect.

**ECG Summary**
- **Rate:** < 60 (usually 40-59)
- **Rhythm:** R- R =
- **P Waves:** Upright (except AVR); similar
- **P-R interval:** 0.12 - 0.20 seconds & consistent
- **qRs:** 0.04 – 0.10 seconds
- **P to qRs:** 1P:1qRs

**Causes:**
- Healthy Athlete
- Sinus Node Disease
- Sleep
- Medications
- Increased Vagal Tone
- Increased Intracranial Pressure
- Valsalva maneuver, Vomiting, Suctioning

**Therapy:**
- Stable – Assess patient; notify physician if new rhythm; 12-lead ECG; observe
- Unstable – Notify physician, 12-lead ECG, support ABCs/determine need for oxygen, confirm or initiate IV, Monitor, Atropine 0.5mg IVP, pacemaker (see page 35 for unstable parameters)
- See OSUMC or unit-specific policy regarding oxygen administration
Sinus Dysrhythmia (Sinus Arrhythmia)

Sinus Dysrhythmia is a normal variation of Normal Sinus Rhythm and in most cases is considered a normal rhythm. The rhythm has slight irregularities. A rhythm strip may appear normal but when measured, the P-P and R-R intervals will reveal a variation of the rhythm (to be considered irregular the R-R must vary more than 0.12 seconds). The most common type of Sinus Dysrhythmia is found in children and is usually associated with respiration; that is the rate speeds up with inspiration and slows with expiration. There are also non-respiratory versions. Some Sinus Dysrhythmias may be bradycardic, if so then the rhythm name would be Sinus Bradydysrhythmia. (A tachycardic rate would be called Sinus Tachydysrhythmia).

It is important to differentiate Sinus Dysrhythmia from a Normal Sinus Rhythm with ectopy. With Sinus Dysrhythmia, all the complexes and waveforms are identical because they originate in the same focus (the SA Node). Ectopic complexes will differ in appearance from the underlying rhythm which is how you know they came from a different focus (atrial tissue, AV Junction, etc.).

Unstable

Four basic parameters classify a patient as unstable. They are:

- **Hypotension** – Systolic blood pressure ↓ 90 mmHg, signs of shock
- **Altered mental status**
- **Shortness of breath** – pulmonary congestion
- **Ischemic chest pain**

**ECG Summary**

- **Rate**: 60 – 100 (may also be < 60 or > 100)
- **Rhythm**: R - R ≠ ( > 0.12 seconds difference)
- **P Waves**: Upright (except AVR); similar
- **P-R interval**: 0.12 - 0.20 seconds & consistent
- **qRs**: 0.04 – 0.10 seconds
- **P to qRs**: 1P:1qRs

**Causes:**
Sinus Dysrhythmia

- Normal in children, young adults & the elderly
- Coronary Artery Disease
- Medications

**Treatment:**
- Assess patient to determine stability; 12-lead ECG, notify physician if new rhythm or unstable
- Generally no treatment is required. If the patient is unstable, it is due to a bradycardic rate

![Sinus Dysrhythmia diagram]

Sinus Pause

Occasionally, the SA Node will fail momentarily and will not initiate an impulse. This might be due to increased vagal stimulation, pharyngeal irritation, carotid sinus massage or deep inspiration. In the case of severely delayed or even permanent cessation, alternative or secondary pacemakers may takeover and maintain the heart rhythm. Two rhythms fall under the Sinus Pause category: Sinus Arrest and Sinus Exit Block.

Sinus Arrest

Sinus Arrest is a failure of the SA Node to initiate an impulse. This is a disorder of automaticity. The rhythm will be slightly irregular as the SA Node will not resume “on time” after the arrest.

Sinus Exit Block
In Sinus Exit Block, the SA Node initiates impulses in a regular rhythm; however, some of the impulses are blocked as they leave the SA Node and therefore do not reach the atria. This is a disorder of conductivity. This rhythm will have P waves that “march out”.

Clinically, it is not necessary to distinguish between a Sinus Arrest and a Sinus Exit Block. If the underlying rhythm is irregular (Sinus Dysrhythmia) then differentiation is impossible, and the general term of Sinus Pause is used.

<table>
<thead>
<tr>
<th>Sinus Pause ECG Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rate:</strong> Usually &lt; 60 due to the pause</td>
</tr>
<tr>
<td><strong>Rhythm:</strong> R- R ≠ due to the pause</td>
</tr>
<tr>
<td><strong>P Waves:</strong> Upright (except AVR); similar</td>
</tr>
<tr>
<td><strong>P-R interval:</strong> 0.12 -0 .20 seconds &amp; consistent</td>
</tr>
<tr>
<td><strong>qRs:</strong> 0.04 – 0.10 seconds</td>
</tr>
<tr>
<td><strong>P to qRs:</strong> 1P:1qRs</td>
</tr>
</tbody>
</table>

**Causes:**
- Vagal Stimulation – overeating, caffeine, nicotine
- Pharyngeal irritation -Intubation
- Valsalva maneuver - Carotid Sinus Massage, deep inspiration
- Sick Sinus Node
- Medications
- Coronary Artery Disease, Myocardial Infarction

**Treatment:**
- Stable – Assess patient to determine stability; 12-lead ECG, determine need for oxygen, ensure adequate IV access, notify physician if new, observe
- Unstable – Ensure ABCs/determine need for oxygen; notify physician; initiate or confirm IV access, Atropine 0.5mg IVP; pacemaker
It is imperative to measure and document the length of the pause and assess the patient’s tolerance of the pause. Does the patient complain of dizziness, shortness of breath or chest pain? Is the blood pressure stable? With any dysrhythmia, the nurse needs to begin to assess the possible causes of the rhythm disturbance and have that information ready to discuss with the physician and health-care team. As you will see, there are several common causes of the various dysrhythmias such as hypoxia, cardiac ischemia, electrolyte imbalance and acidosis just to name a few. It is pointless to simply medicate for the dysrhythmia if the underlying cause is not corrected.

**Clinical Picture**

It is just as imperative for the nurse or healthcare provider to notice and document trends in the clinical picture. Trends in vital signs, physical assessments or patient complaints can assist the healthcare provider in anticipating untoward clinical events. When these signs are not recognized, this is known as “Failure to Rescue”. It has been shown that patients will exhibit signs and symptoms as far out as 24 to 48 hours prior to full cardiac arrest. The ECG is just one tool that, if used correctly, can provide clues to the patient’s underlying disease process or injury.

![Normal Sinus Rhythm with a 2.44 Second Sinus Pause](image)
Electrocardiographic Artifact

Electrocardiographic artifact or electrical noise can be seen on the ECG strip as a result of patient body movement, muscle tremor, electrical current near the patient, or poor skin-to-electrode contact. It is important to recognize artifact as such and not to confuse it with other rhythms such as Atrial Fibrillation or Ventricular Tachycardia.

![Normal Sinus Rhythm with Artifact](image)

Documentation & Housekeeping

The monitor ECG strip should be placed in the Nursing and Allied Health Professions Notes. A note immediately following the strip should include the patient’s name, date, time, rhythm interpretation, heart rate, PR interval, qRs interval, QT interval, any wave abnormalities and RN signature. Rhythm interpretation documentation should include a full description such as “Normal Sinus Rhythm with 2.44 second Sinus Pause”. Do not write directly on the rhythm strip! Rhythm strips should be obtained at least every 8 hours (or per unit-specific policy) and with any significant change in rhythm. Monitor recall should be done per unit policy. The alarm limits should be set at 50 – 150 bpm (unless physician order dictates otherwise), and telemetry batteries should be changed q24 hours (Policy: ECG Monitoring, Continuous).
Bradycardia

Assess patient to determine stability
Stable or Unstable?

Stable

Yes

Notify MD if new rhythm
12-lead ECG
Ensure IV access
Observe and Reassess

No

 Ensure ABCs/determine need for oxygen
 Notify MD
 Initiate or confirm IV access (at least 2 sites)
 Remain with patient and continually reassess

Unstable

• Atropine 0.5mg IVP (total dose of 3mg)
• TCP or Transvenous pacer
• Dopamine or Epinephrine drips (Hypotension)

Sinus Rhythms Causes and Treatments
<table>
<thead>
<tr>
<th>Rhythm</th>
<th>Causes</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus Tachycardia</td>
<td>♦ Exercise</td>
<td>♦ Treat or remove cause</td>
</tr>
<tr>
<td></td>
<td>♦ ↑ Temperature</td>
<td>♦ MI – Beta Blockers</td>
</tr>
<tr>
<td></td>
<td>♦ Hypovolemia, Dehydration</td>
<td></td>
</tr>
<tr>
<td></td>
<td>♦ Stress, Anxiety, Fight or Flight</td>
<td></td>
</tr>
<tr>
<td></td>
<td>♦ Hypoxia, CHF, Acute MI</td>
<td></td>
</tr>
<tr>
<td></td>
<td>♦ Sympathetic Stimulation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>♦ Meds or Substances</td>
<td></td>
</tr>
<tr>
<td>Sinus Bradycardia</td>
<td>♦ Normal</td>
<td>♦ Stable – Notify MD if new rhythm; 12-lead ECG; Observe &amp; Reassess</td>
</tr>
<tr>
<td></td>
<td>♦ Increased Vagal Tone</td>
<td>♦ Unstable</td>
</tr>
<tr>
<td></td>
<td>♦ Medications</td>
<td>♦ Notify MD</td>
</tr>
<tr>
<td></td>
<td>♦ Diseased SA Node</td>
<td>♦ Ensure ABCs/determine need for oxygen</td>
</tr>
<tr>
<td></td>
<td>♦ Hypothermia</td>
<td>♦ Ensure IV Access</td>
</tr>
<tr>
<td></td>
<td></td>
<td>♦ Atropine 0.5mg IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>♦ Pacemaker</td>
</tr>
<tr>
<td>Sinus Dysrhythmia</td>
<td>♦ Normal</td>
<td>♦ Assess</td>
</tr>
<tr>
<td></td>
<td>♦ Coronary Artery Disease</td>
<td>♦ Notify MD</td>
</tr>
<tr>
<td></td>
<td>♦ Myocardial Infarction</td>
<td></td>
</tr>
<tr>
<td></td>
<td>♦ Medications</td>
<td></td>
</tr>
<tr>
<td>Sinus Pause (Arrest)</td>
<td>♦ Increased Vagal Tone</td>
<td>♦ Stable – Notify MD if new rhythm; 12-lead ECG; Observe &amp; Reassess</td>
</tr>
<tr>
<td></td>
<td>♦ Coronary Artery Disease</td>
<td>♦ Unstable</td>
</tr>
<tr>
<td></td>
<td>♦ Rheumatic Heart Disease</td>
<td>♦ Notify MD</td>
</tr>
<tr>
<td></td>
<td>♦ Myocarditis</td>
<td>♦ Ensure ABCs</td>
</tr>
<tr>
<td></td>
<td>♦ Medications</td>
<td>♦ O2 at 2L/NC</td>
</tr>
<tr>
<td></td>
<td></td>
<td>♦ Ensure IV Access</td>
</tr>
<tr>
<td></td>
<td></td>
<td>♦ Atropine 0.5mg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>♦ Pacemaker</td>
</tr>
</tbody>
</table>
Pacemaker Functions

Pacemakers, whether external/internal or temporary/permanent, have three basic functions:

- Firing
- Capturing
- Sensing
Pacemaker Function: Firing

- **Firing:** The pacemaker discharges the electricity into the myocardium
  
  - Delivery of preset milliamps (mA)
  
  - The programmed pacemaker rate determines how fast the electricity is delivered
  
  - When the pacemaker has properly fired, you will notice a pacemaker spike
    - Thin, straight line (vertical) on the tracing
Pacer Spikes = Pacer Firing

Since we see the thin pacemaker spike, we know that this pacemaker has appropriately fired, or discharged electricity into the myocardium.
Pacemaker Function: Capture

**Capture**

- Pacing stimulus delivers enough amperes (mAs) to depolarize the myocardium
- mAs must be above the electrical threshold of the myocardium to cause depolarization

- **Ventricular capture** = wide bizarre qRs that immediately follows the pacer spike

- **Atrial capture** = normal complex with P/qRs/T waves that immediately follows the pacer spike

- **Remember**: Only shows electrical capture – does not mean that mechanical capture (or contraction) has occurred
Pacer Firing and capturing 1:1

The pacer spikes show that the pacer fired; the wide complex qRs shows ventricular capture.
Pacemaker Function: Sensing

**Sensing**: Pacemaker is capable of “seeing” or sensing the patient’s own inherent beats

- This inhibits the pacemaker from firing when the patient is having an inherent beat
- Allows pacer to fire on demand when the patients own rate drops below the preset pacer rate
- If pacemaker set on “Fixed” mode, no sensing occurs
Failure Of Pacemaker Function

- Pacemakers can fail in any one or more of their necessary functions.

- Failure includes:
  - Failure to fire
  - Failure to capture
  - Failure to sense
Pacemaker Failure: Failure to Fire

- **Failure to fire**
  - No pacer spikes noted when inherent rate drops below the pacer’s programmed rate

- **Causes:**
  - Malfunction of the pulse generator
  - Low battery
  - Wire fracture
  - Loose connections or other mechanical problems with the pacemaker
Failure to Fire

Pacemaker fires (spike) and captures (wide-complex qRs following the spike).

Absence of a pacer spike = Failure to Fire
Pacemaker Failure: Failure to Capture

◆ Failure to capture: The pacemaker stimulus does not “capture” or depolarize the ventricle.
  ◆ No complex noted after a pacer spike (no “P” wave after an atrial spike nor a wide bizarre “QRS” after a ventricular spike)

◆ Cause:
  ◆ Displaced tip
  ◆ Increased ischemia
  ◆ Loose connections
Failure to Capture

In this tracing, you see a sudden loss of ventricular capture. The pacer has fired appropriately as evidenced by the pacer spikes; however, the ventricles were not “captured” with these two spikes.
Pacemaker Failure: Failure to Sense

◆ **Failure to Sense**: Pacemaker does not “see” the patients own inherent rhythm and continues to fire
  - Pacer spikes are noted in the middle of an inherent rhythm
  - Can be life threatening if spike is fired on a “T” wave when ventricles are repolarizing resulting in ventricular tachycardia or ventricular fibrillation
  - Pacemaker competition occurs

◆ **Cause:**
  - Poor lead placement
  - Loose wires or connections
  - Low battery
  - Circuitry malfunction
In this tracing, you can see pacer spikes falling throughout the patient’s own inherent cardiac rhythm. The pacer did not “see” this inherent rhythm and continued to fire even though it was not needed.
<table>
<thead>
<tr>
<th>Rhythm</th>
<th>P waves</th>
<th>qRs</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Sinus Rhythm (NSR)</td>
<td>Round, upright, similar PRI: 0.12-0.20</td>
<td>0.04-0.10</td>
<td>Rate: 60-100 bpm</td>
</tr>
<tr>
<td></td>
<td>P:qRs 1:1</td>
<td></td>
<td>Rhythm: R</td>
</tr>
<tr>
<td>Sinus Tachycardia</td>
<td>Round, upright, similar PRI: 0.12-0.20</td>
<td>0.04-0.10</td>
<td>101-150 bpm</td>
</tr>
<tr>
<td></td>
<td>P:qRs 1:1</td>
<td></td>
<td>Rhythm: R</td>
</tr>
<tr>
<td>Sinus Bradycardia</td>
<td>Round, upright, similar PRI: 0.12-0.20</td>
<td>0.04-0.10</td>
<td>≤ 59 bpm</td>
</tr>
<tr>
<td></td>
<td>P:qRs 1:1</td>
<td></td>
<td>Rhythm: R</td>
</tr>
<tr>
<td>Sinus Dysrhythmia</td>
<td>Round, upright, similar PRI: 0.12-0.20</td>
<td>0.04-0.10</td>
<td>Rate: variable</td>
</tr>
<tr>
<td></td>
<td>P:qRs 1:1</td>
<td></td>
<td>Rhythm: I</td>
</tr>
<tr>
<td>PAC</td>
<td>Varies from sinus Ps; small, peaked or notched; may be buried in preceding sinus T wave; PRI may be different than sinus PRI</td>
<td>0.04-0.10</td>
<td>Rate: Variable</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Rhythm: I (d/t PAC)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Timing: Early</td>
</tr>
<tr>
<td>Rhythm</td>
<td>P waves</td>
<td>qRs</td>
<td>Notes</td>
</tr>
<tr>
<td>---------------------------------------</td>
<td>--------------------------------</td>
<td>--------</td>
<td>--------------------------------------------</td>
</tr>
<tr>
<td><strong>Supraventricular Tachycardia (SVT)</strong></td>
<td>Not visible</td>
<td>0.04-0.10</td>
<td>Rate: ≥ 150 bpm</td>
</tr>
<tr>
<td></td>
<td>PRI: N/A</td>
<td></td>
<td>Rhythm: R</td>
</tr>
<tr>
<td><strong>Atrial Flutter</strong></td>
<td>N/A “F” or “flutter” waves</td>
<td>0.04-0.10</td>
<td>Rate: Variable</td>
</tr>
<tr>
<td></td>
<td>PRI: N/A</td>
<td></td>
<td>Rhythm: R or I</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Saw tooth pattern</td>
</tr>
<tr>
<td><strong>Atrial Fibrillation</strong></td>
<td>N/A Fibrillating baseline</td>
<td>0.04-0.10</td>
<td>Rate: Varies</td>
</tr>
<tr>
<td></td>
<td>PRI: N/A</td>
<td></td>
<td>Rhythm: I</td>
</tr>
<tr>
<td><strong>PJC</strong></td>
<td>No P wave, or inverted P wave</td>
<td>0.04-0.10</td>
<td>Rate: Varies</td>
</tr>
<tr>
<td></td>
<td>before or after qRs</td>
<td></td>
<td>Rhythm: I (d/t PJC)</td>
</tr>
<tr>
<td></td>
<td>PRI: ≤0.12 if present before qRs</td>
<td></td>
<td>Timing: Early</td>
</tr>
<tr>
<td><strong>Junctional escape Rhythm</strong></td>
<td>No P wave, or inverted P wave</td>
<td>0.04-0.10</td>
<td>Rate: 40-60 bpm</td>
</tr>
<tr>
<td></td>
<td>before or after qRs</td>
<td></td>
<td>Rhythm: R</td>
</tr>
<tr>
<td>Rhythm</td>
<td>P waves</td>
<td>qRs</td>
<td>Notes</td>
</tr>
<tr>
<td>------------------------</td>
<td>----------------------------------------------</td>
<td>--------</td>
<td>--------------------------------------------</td>
</tr>
<tr>
<td><strong>Accelerated Junctional</strong></td>
<td>No P wave, or inverted P wave before or after qRs</td>
<td>0.04-0.10</td>
<td>Rate: 61-100 bpm  &lt;br&gt; Rhythm: R</td>
</tr>
<tr>
<td></td>
<td>PRI: ≤0.12 if present before qRs</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Junctional Tachycardia</strong></td>
<td>No P wave, or inverted P wave before or after qRs</td>
<td>0.04-0.10</td>
<td>Rate: 101-150 bpm</td>
</tr>
<tr>
<td></td>
<td>PRI: ≤0.12 if present before qRs</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>PVC</strong></td>
<td>None</td>
<td>&gt;0.12</td>
<td>Rate: Variable  &lt;br&gt; Rhythm: I (d/t PVC)  &lt;br&gt; Timing: Early  &lt;br&gt; Pattern: Bigeminy-every other beat; Trigeminy-every third beat; Multiformed-different shapes; Couplelet-two in a row</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rhythm</td>
<td>P waves</td>
<td>qRs</td>
<td>Notes</td>
</tr>
<tr>
<td>---------------------------</td>
<td>---------</td>
<td>------</td>
<td>--------------------</td>
</tr>
<tr>
<td><strong>Idioventricular Rhythm</strong></td>
<td>None</td>
<td>&gt;0.12</td>
<td>Rate: 20-40 bpm</td>
</tr>
<tr>
<td></td>
<td>PRI: N/A</td>
<td></td>
<td>Rhythm: R</td>
</tr>
<tr>
<td><strong>Accelerated Idioventricular</strong></td>
<td>None</td>
<td>&gt;0.12</td>
<td>Rate: 41-100 bpm</td>
</tr>
<tr>
<td></td>
<td>PRI: N/A</td>
<td></td>
<td>Rhythm: R</td>
</tr>
<tr>
<td><strong>Ventricular Tachycardia</strong></td>
<td>None</td>
<td>&gt;0.12</td>
<td>Rate: &gt;100 bpm</td>
</tr>
<tr>
<td></td>
<td>PRI: N/A</td>
<td></td>
<td>Rhythm: R</td>
</tr>
<tr>
<td><strong>Ventricular Fibrillation</strong></td>
<td>None</td>
<td>N/A</td>
<td>Rate: N/A</td>
</tr>
<tr>
<td></td>
<td>PRI: N/A</td>
<td></td>
<td>Rhythm: Chaos</td>
</tr>
<tr>
<td>Rhythm</td>
<td>P waves</td>
<td>qRs</td>
<td>Notes</td>
</tr>
<tr>
<td>---------------------------------------------</td>
<td>----------------------------------------------</td>
<td>-------</td>
<td>--------------------------------------------</td>
</tr>
<tr>
<td><strong>Asystole</strong></td>
<td>May be present; round, upright &amp; similar</td>
<td>N/A</td>
<td>Rate: 0</td>
</tr>
<tr>
<td></td>
<td>PRI: N/A</td>
<td></td>
<td>Rhythm: R (if P waves present)</td>
</tr>
</tbody>
</table>

- **Any organized rhythm that should perfuse but patient is PULSELESS = “Rhythm Name with PEA”**

- **1st degree AV Block**
  - Round, upright, similar
  - PRI: >0.20 but <0.40
  - P:qRs 1:1
  - 0.04-0.10
  - Only block that does not drop qRs
  - Consistent PRI

- **2nd degree AV Block, Type II**
  - Round, upright, similar
  - PRI: normal or long; consistent across strip
  - #P > #qRs
  - 0.04-0.10 OR ≥ 0.12
  - Consistent PRI when P conducts to qRs; unpredictable drop of qRs
<table>
<thead>
<tr>
<th>Rhythm</th>
<th>P waves</th>
<th>qRs</th>
<th>Notes</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>2nd degree AV Block, Type I</td>
<td>Round, upright, similar</td>
<td>0.04-0.10</td>
<td>Cyclic pattern</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PRI: progressive lengthening</td>
<td></td>
<td>Inconsistent PRI</td>
<td></td>
</tr>
<tr>
<td></td>
<td>until drop of qRs</td>
<td></td>
<td>Predictable drop of qRs after</td>
<td></td>
</tr>
<tr>
<td></td>
<td>#P &gt; #qRs</td>
<td></td>
<td>lengthening PRI</td>
<td></td>
</tr>
<tr>
<td>3rd degree AV Block</td>
<td>Round, upright, similar</td>
<td>0.04-0.10</td>
<td>Inconsistent PRI</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PRI: N/A</td>
<td>OR ≥0.12</td>
<td>Ps “march out”</td>
<td></td>
</tr>
<tr>
<td></td>
<td>#P &gt; #qRs</td>
<td></td>
<td>No relationship between P and</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ps “march out”</td>
<td></td>
<td>qRs</td>
<td></td>
</tr>
</tbody>
</table>
## ECG Rhythm Characteristics

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Defib</th>
<th>Synch Cardiovert</th>
<th>TCP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adenosine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epinephrine</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
CONDUCTION SYSTEM IN RELATION TO HEART STRUCTURES

- **SA Node**
  - Intrinsic Rate: 60-100

- **AV Junction**
  - Intrinsic Rate: 40-60

- **Atrial Rhythms**

- **Ventricular Rhythm**
  - Intrinsic Rate: 20-40
Sinus Rhythms

The SA Node is the only place that gives you the perfect, “normal” sinus rhythm. The rhythms are characterized by round, upright, similar-appearing P waves, followed by a normal PR Interval. There is one narrow QRS that follows each P wave. The rhythms may differ in their rate (≤60 bpm to between 100-150 bpm). All other characteristics will remain the same.

Junctional Rhythms

The AV Junction or Node is able to take over as an “escape” pacemaker for the heart in the absence of a functioning SA Node. Since the junction lies superior to the ventricles, the rhythms maintain a narrow QRS complex, and are regular. However, Junctional rhythms are differentiated from SA Node rhythms by the P wave. The P wave may have one of three characteristics: absent; inverted before the QRS; inverted following the QRS. The inherent rate of the junction is 40-60 bpm. Again, the Junctional rhythms may differ in their rate (40-60 bpm, 60-100 bpm or 100-150 bpm), but all other characteristics will remain the same.

Ventricular Rhythms

The ventricular myocardium is able to take over as an “escape” pacemaker for the heart in the absence of upper-level functioning pacemakers. Since the ventricles deliver their impulses across the myocardium (no intact pathways), the QRS is wide and bizarre in appearance. There are no associated P waves (the P waves only come from superior to the ventricles, and since the impulse is initiated in the ventricles, no P waves are present). Ventricular rhythms are hallmarked by the wide, bizarre-appearing QRS complex. The inherent rate of the ventricles is 20-40 bpm. Again, the ventricular rhythms may differ in their rate (20-40 bpm, 40-100 bpm, 100-220 bpm), but all other characteristics will remain the same.

Ventricular Fibrillation is a chaotic rhythm that does not produce a pulse and is not compatible with life. The heart is simply “quivering”, and does not produce an organized contraction. The patient in “V Fib” requires immediate life support measures.

Ventricular Asystole is a rhythm that does not produce a pulse and is not compatible with life. The heart is motionless, and does not produce a contraction. There may be P waves on the rhythm strip, but this signifies atrial depolarization, which does not produce a pulse. The patient in “Asystole” requires immediate life support measures.
PAC (Premature Atrial Complex)

A PAC is a complex that falls early in the cardiac cycle. It is produced by an ectopic site that fired from the atrial myocardium. It is similar in appearance to a normal sinus complex, but it will be identified in one of three ways: 1. There will be a change in the P wave (the PAC's P wave may be smaller or more peaked than the other “normal” sinus P waves on the rhythm strip). 2. There will be a change in the PR Interval (the PAC’s PR interval will be different from the “normal” sinus PR intervals across the rhythm strip). 3. There will be a change in the T waves (the T wave of the preceding sinus complex before the PAC will be larger in size than all other sinus complex T waves-this shows a buried P wave inside the sinus complex T wave).

PJC (Premature Junctional Complex)

A PJC is a complex that falls early in the cardiac cycle. It is produced by an ectopic site that fired from the AV Junction or Node. It maintains the fundamental characteristics of a Junctional complex (narrow QRS and either no P wave, an inverted P wave before the QRS or an inverted P wave following the QRS).

PVC (Premature Ventricular Complex)

A PVC is a complex that falls early in the cardiac cycle. It is produced by an ectopic site that fired from the ventricular myocardium. It maintains the fundamental characteristics of a Ventricular complex (no P wave and a wide, bizarre-appearing QRS). PVCs are described according to their number or pattern on the rhythm strip (rare, occasional, frequent; bigeminal or trigeminal) and their shape (uniformed or multiformed). It is important to thoroughly describe PVCs as their shape, number and pattern become clinically significant (increasing numbers of PVCs demonstrate ventricular instability, and PVCs of different shapes demonstrate multiple ectopic sites that are firing from the ventricles).

SVT (Supraventricular Tachycardia)

SVT is a name assigned to a group of rhythms that originate from above the ventricles (“supraventricular”), with a rate of ≥150 bpm (tachycardia), and are regular. At this rate, it is not possible to accurately determine if a P wave is present, however, the clinician knows that this rhythm originated above the ventricles due to the narrow QRS complex. Until the rhythm is slowed via medical interventions, the clinician is unable to determine the exact origin of the tachycardia (sinus, atrial or Junctional).
**Atrial Flutter**

Atrial Flutter is a rhythm that originates in the atrial myocardium, at rates between 250-350 bpm. The atrial rate is regular, and instead of P waves, F waves are present. F waves create a baseline that resembles a picket fence or the blades of a saw (saw tooth pattern). The AV Node will “filter” the atrial impulses to the ventricles, so that not all impulses reach the ventricles to cause contraction. The “filtering” can be consistent or inconsistent (every 3rd atrial beat is allowed through, versus a random pattern of beats allowed through), which is what makes the overall rhythm regular or irregular. Since the rhythm originates above the ventricles, the QRS complex is narrow.

**Atrial Fibrillation**

Atrial Fibrillation is a rhythm that originates in the atrial myocardium, at rates greater than 350 bpm. At this rate, no discernible waves are able to be seen, so a “wavy” or “fibrillating” baseline is present. The AV Node cannot “filter” atrial beats with any regularity at these rates, so the overall rhythm is always irregular. Since the rhythm originates above the ventricles, the QRS complex is narrow.

**1st Degree AV Block**

First Degree AV Block is similar in appearance to a “normal” sinus rhythm. The key difference is a prolonged PR Interval. The impulse leaves the SA Node, but is slowed at the AV Node, creating a prolonged PR Interval. Once the impulse passes the AV Node, conduction proceeds normally. Since the rhythm originates in the SA Node, there are upright, similar “normal” sinus P waves, and a narrow QRS complex that follows each P wave.

**2nd Degree AV Block Type 2**

Second Degree AV Block Type 2 is characterized by P waves that conduct to QRS complexes, but without warning, the atrial impulse is not conducted through to the ventricles, and a QRS complex is dropped. The PR Interval of the conducted beats is usually normal, but may be prolonged. In either case, the PR Interval is consistent across the rhythm strip. The QRS complexes may be narrow if the escape pacemaker is in the Junction, or wide if the escape pacemaker is in the ventricular myocardium. The hallmark of a Second Degree AV Block Type 2 is that when the P wave is conducted to the QRS, the PR Interval is consistent; when the QRS complex is dropped, it is dropped without a change to the PR Intervals.
2nd Degree AV Block Type 1

Second Degree AV Block Type 1 is characterized by a progressive lengthening of the PR Interval until a QRS complex is dropped. This is a cyclic pattern, which regularly repeats throughout the rhythm strip. Each cycle is identical to the others, and would match up perfectly if placed atop one another. This rhythm is usually transient.

3rd Degree AV Block

Third Degree AV Block is a complete lack of communication between the atria and ventricles. The atria will initiate regular impulses that are not conducted through to the ventricles. The P waves are upright and similar throughout the rhythm strip. Since there is no communication between the atria and ventricles, there is no PR Interval. The QRS complexes may be narrow if the escape pacemaker is in the Junction, or wide if the escape pacemaker is in the ventricular myocardium. The heart rate is also dependent upon the location of the escape pacemaker (other factors may play a role as well). The overall rhythm is irregular, but taken individually, the atrial and the Junctional or ventricular impulses are regular and “march out”.