Telemetry Interpretation

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Purpose
The purpose of this course is to provide the healthcare professional the tools to perform basic electrocardiogram (ECG) interpretation. This course is designed for the healthcare professional who has limited or no previous ECG experience. Key basic rhythms will be taught along with nursing priorities and initial treatment strategies for each rhythm. To facilitate learning, a systematic approach for interpretation will be used throughout this course.

You need limited supplies and tools to make this learning opportunity a success. To enhance ECG strip analysis we recommend you purchase hand calipers to determine heart rate, regularity, and all ECG intervals. If you do not own calipers already, they can typically be purchased for approximately $10.00 at most medical book and uniform stores.

Objectives
After successful completion of this continuing education self-study course, participants will be able to:

1. Describe the flow of blood through the normal heart and lungs.
2. State the differences between electrical and mechanical cells within the heart.
3. Describe the location and function of the following structures:
   - Sinoatrial (SA node)
   - Atrioventricular (AV) junction
   - Bundle of His
   - Bundle branches
   - Purkinje fibers
4. Name the three cardiac pacemakers, their locations and intrinsic heart rates of pacing.
5. Describe the correct electrode placement for leads I, II, III and MCL1 or V1.
6. Define and describe the significance of:
   - P wave
   - PR interval
   - QRS complex
   - ST segment
   - T wave
   - QRS interval
7. State one method of heart rate calculation.
8. Describe ECG characteristics, nursing priorities, and initial treatment options for the following rhythms:
   - Normal sinus rhythm
   - Sinus bradycardia
Introduction
Interpretation of ECGs (electrocardiograms; also known as EKGs) is one of the building blocks of critical care nursing. Before the actual ECG interpretation can occur, a significant base of cardiac knowledge must be built. This groundwork will include the topics such as the normal conduction system, electrode placement, lead selection, ECG paper, and specific ECG waveforms.

Because this information is complex and potentially confusing, you might want to re-read the sections you find more challenging. You do not need to finish this course in one sitting. The course encourages you to stop and practice at your own pace. Throughout this course, you will be asked to participate in "Question and Answer" sections, along with actual ECG strip practice. This is to help you gauge your progress. For continuing education credits, the post test at the conclusion of this course must be completed. Enjoy your ECG adventure!

IMPORTANT NOTE:
This is NOT a course that can be done in one sitting or with only one reading. Be prepared to spend some time and review the materials more than once in order to learn the material and pass the post test.

Section 1: Anatomy and Physiology

Heart Chambers and Normal Blood Flow
The heart is a four-chambered structure made up of two receiving chambers called atria and two pumping chambers called ventricles. The right atrium receives oxygen-depleted blood returning from the body through the superior and inferior vena cava. The right ventricle pushes the oxygen poor blood to the lungs through the pulmonary arteries. The blood is oxygenated in the lungs. The left atrium receives oxygen-rich blood returning from the lungs through pulmonary veins. The left ventricle pushes the oxygen-rich blood out through the aorta, which directs the blood to all parts of the body. The left ventricle is a high-pressure chamber that is approximately three times thicker than the right ventricle. The right and left atria and ventricular chambers are separated by a septal wall or septum (Jarvis, 2011).
Section 1: Anatomy and Physiology
Heart Valves

When blood flows through the heart, it follows a unidirectional pattern. There are four different valves within the myocardium and their functions are to assure blood flows from the right to left side of the heart and always in a “forward” direction.

The two valves found between the atria and ventricles are appropriately called atrioventricular (A-V) valves. The **tricuspid valve** separates the right atrium from the right ventricle. Similarly, the **mitral valve** separates the left atrium from the left ventricle.

The two remaining valves are called semilunar valves (because they look like half-moons). The valve located where the pulmonary artery meets the right ventricle is called the **pulmonic valve**. The **aortic valve** is located at the juncture of the left ventricle and aorta. Both semilunar valves prevent backflow of blood into the ventricles.

<table>
<thead>
<tr>
<th>Valve Type</th>
<th>Valve Name</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrioventricular (AV)</td>
<td>Tricuspid</td>
<td>Separates right atrium and right ventricle</td>
</tr>
<tr>
<td></td>
<td>Mitral</td>
<td>Separates left atrium and left ventricle</td>
</tr>
<tr>
<td>Semilunar</td>
<td>Pulmonic</td>
<td>Between right ventricle and pulmonary artery</td>
</tr>
<tr>
<td></td>
<td>Aortic</td>
<td>Between left ventricle and aorta</td>
</tr>
</tbody>
</table>

The left ventricle is three times thicker than the right ventricle.
Section 1: Anatomy and Physiology
Correlation to Heart Sounds

The first heart sound called S1 (or “Lub” of the “Lub-Dub” sound) is the result of closure of the tricuspid and mitral valves during ventricular contraction. The second heart sound called S2 (or “Dub”) occurs at the end of ventricular contraction due to the closure of the aortic and pulmonic valves.

Atrial Kick

About 2/3 of the atrial blood flows passively from the atria into the ventricles. When atrial contraction occurs (and the AV valves are open), the atrial blood is pushed down into the ventricles. This atrial contribution is called atrial kick and accounts for approximately 30% of the cardiac output (the amount of blood ejected by the left ventricle into the aorta in one minute) (Heitman, Siroky, Noble, & Thomason, 2012). This concept will be discussed in detail, as it relates to specific rhythms, later in this module.

Test Yourself

The valve that separates the left atrium from the left ventricle is the:

1. Tricuspid valve
2. Mitral valve - correct
3. Aortic valve

Section 2: Basic Electrophysiology

To understand and interpret ECG rhythms it is necessary for you to understand the electrical activity which is occurring within the heart. The term electrocardiography literally means the recording of the electrical activity of the heart muscle.
Electrical and Mechanical Properties

Two distinct components must occur for the heart to be able to contract and pump blood. These components are A) an electrical impulse and B) a mechanical response to the impulse.

- The **electrical** impulse tells the heart to beat. This property is called **automaticity**. Automaticity means that these specialized cells within the heart can discharge an electrical current without an external pacemaker, or stimulus from the brain via the spinal cord.
- The **mechanical** beating or contraction of the heart occurs after the electrical stimulation. When the mechanical contraction occurs, the person will have both a heart rate and a blood pressure.

In summary, the heart has both an electrical and a mechanical property (Heitman et al., 2012; Jacobson, Marzlin, & Webner, 2014).

Section 2: Basic Electrophysiology

Heart Cells

The heart also has two distinct types of cells. There are **electrical (conductive) cells**, which initiate electrical activity and conduct it through the heart. There are also **mechanical (contracting) cells**, which respond to the electrical stimulus and contract to pump blood.

The contracting or myocardial “working cells” contain contractile filaments. When these cells are electrically stimulated, these filaments slide together and the myocardial cell contracts and the atria or ventricular chambers contract. This is how we get our pulse and blood pressure.

An ECG tracing is designed to give a graphic display of the electrical activity in the heart. The pattern displayed on the ECG is called the heart rhythm. However, an ECG cannot tell you about the mechanical activity of the heart. You will have to assess the patient's blood pressure and pulse to determine this (Jacobsen et al., 2014).

Section 2: Basic Electrophysiology

Depolarization and Repolarization

In a cardiac cell, two primary chemicals provide the electrical charges: **sodium** (Na+) and **potassium** (K+). In the resting cell, the potassium is mostly on the inside, while the sodium is mostly on the outside. This results in a **negatively charged cell at rest** (the interior of the cardiac cell is mostly negative or **polarized** at rest). When **depolarized**, the interior cell becomes positively charged and the cardiac cell will contract.

In summary, the polarized or resting cell will carry a negative charge on the inside. When depolarized, the opposite will occur. This is due to the movement of sodium and potassium across the cell membrane. Depolarization moves a wave through the myocardium. As the wave of depolarization stimulates the heart’s cells, they become positive and begin to contract. This cell-to-cell conduction of depolarization through the myocardium is carried by the fast moving sodium ions.
Repolarization is the return of electrical charges to their original state. This process must happen before the cells can be ready to conduct again. Even though we have not yet introduced the ECG waveforms to you, simply look at the diagram below and note the depolarization and repolarization phases as they are represented on the ECG (Jones, 2008).

You are probably wondering how does all of this relate to what is happening in the heart and how is it reflected on the ECG? The electrical cells in the heart are arranged in a system of pathways called the conduction system.

Test Yourself

When the electrical charges in a cell return to their original state, it is known as:
1. Polarization
2. Repolarization - correct
3. Depolarization

Section 3: The Conduction System
The specialized electrical cells in the heart are arranged in a system of pathways called the conduction system. These specialized electrical cells and structures guide the wave of myocardial depolarization.

The physical layout of the conduction system is shown in the picture to the right. It is important that you understand the sequence of events within this conduction system. This is something that you will need to memorize because it will help you to understand normal and abnormal rhythms.

The conduction system consists of the Sinoatrial node (SA node), Atrioventricular Node (AV node), Bundle of His (also called the AV Junction), Right and Left Bundle Branches, and Purkinje Fibers (Jacobsen, et al., 2012; Jarvis, 2011).

Let us now discuss each structure in more detail.

Section 3: The Conduction System
Sinoatrial (SA) Node
The Sinoatrial node (also called the SA node or sinus node) is a group of specialized cells located in the posterior wall of the right atrium. The SA node normally depolarizes or paces more rapidly than any other part of the conduction system. It sets off impulses that trigger atrial depolarization and contraction. Because the SA node discharges impulses quicker than any other part of the heart, it is commonly known as the natural pacemaker of the heart. The SA node normally fires at a rate of 60-100 beats per minute.

After the SA node fires, a wave of cardiac cells begin to depolarize. Depolarization occurs throughout both the right and left atria (similar to the ripple effect when a rock is thrown into a pond). This impulse travels through the atria by way of inter-nodal pathways down to the next structure, which is called the AV node.

Remember the term mentioned before called “atrial kick”? Atrial kick occurs when the atria contract and dump their blood down into the ventricles. This atrial contraction contributes up to 30% of the cardiac output, which is obviously an important element toward maintaining our blood pressure. So remember... the SA node is not only the primary pacemaker of the heart but also triggers atrial depolarization and the contribution of the atrial kick (Heitman et al., 2012; Jacobson, Marzlin, & Webner, 2014).

The heart is truly an amazing organ. Not only does it have one dominant pacemaker (the SA node) it also has two back-up pacemakers. One back-up pacer is located in the area of near the Bundle of His. The final back-up pacer is located in the ventricles along the Purkinje fibers. More interesting information on this later...

Section 3: The Conduction System
Atrioventricular (AV) Node and AV Junction

The next area of conductive tissue along the conduction pathway is at the site of the atrioventricular (AV) node. This node is a cluster of specialized cells located in the lower portion of the right atrium, above the base of the tricuspid valve. The AV node itself possesses no pacemaker cells.

The AV node has two functions. The first function is to DELAY the electrical impulse in order to allow the atria time to contract and complete filling of the ventricles. The second function is to receive an electrical impulse and conduct it down to the ventricles via the AV junction and Bundle of His (Jarvis, 2011).

Section 3: The Conduction System
Bundle of His

After passing through the AV node, the electrical impulse enters the Bundle of His (also referred to as the common bundle). The bundle of His is located in the upper portion of the interventricular septum and connects the AV node with the two bundle branches. If the SA node should become diseased or fail to function properly, the Bundle of His has pacemaker cells, which are capable of discharging at an intrinsic rate of 40-60 beats per minute. This back-up pacemaker function can really come in handy!

The AV node and the bundle of His are referred to collectively as the AV junction. The Bundle of His
conduces the electrical impulse down to the right and left bundle branches.

The right bundle branch spreads the wave of depolarization to the right ventricle. Likewise, the left bundle branch spreads the wave of depolarization to both the interventricular septum and the left ventricle. The left bundle further divides into three branches or fascicles. The bundle branches further divide into **Purkinje fibers** (Heitman et al., 2012; Jacobson, Marzlin, & Webner, 2014).

**Section 3: The Conduction System**

**Purkinje Fibers**

We are now coming to the end of this amazing cardiac conduction system. At the terminal ends of the bundle branches, smaller fibers distribute the electrical impulses to the muscle cells, which stimulate contraction. This web of fibers is called the Purkinje fibers. The Purkinje fibers penetrate about 1/4 to 1/3 of the way into the ventricular muscle mass and then become continuous with the cardiac muscle fibers. The electrical impulse spreads rapidly through the right and left bundle branches and Purkinje fibers to reach the ventricular muscle, causing ventricular contraction, or systole.

These Purkinje fibers within the ventricles also have intrinsic pacemaker ability. This third and final pacemaker site of the myocardium can only **pace at a rate of 20-40 beats per minute** (Heitman et al., 2012; Jacobson, et al., 2014).

You have probably noticed that the further you travel away from the SA node, the slower the backup pacemakers become. As common sense tells you, if you only have a heart rate of 30 (from the ventricular back-up pacemaker), your blood pressure is likely to be low and you might be quite symptomatic.

<table>
<thead>
<tr>
<th>Summary of Pacemaker Function</th>
<th>Pacemaker Hierarchy</th>
<th>Location</th>
<th>Pacing Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 1 (normal)</td>
<td>SA Node</td>
<td></td>
<td>60-100 beats/minute</td>
</tr>
<tr>
<td>Level II (back-up system)</td>
<td>Bundle of His/ AV Node/ Junction</td>
<td></td>
<td>40-60 beats/minute</td>
</tr>
<tr>
<td>Level III (lowest back-up system)</td>
<td>Purkinje Fibers within Ventricles (typically called the Ventricular Pacemaker)</td>
<td></td>
<td>20-40 beats/minute</td>
</tr>
</tbody>
</table>

**Section 3: The Conduction System**

View this video demonstrating the conduction system of the heart in relation to an ECG.

[https://www.youtube.com/watch?v=KWrzdJY4G9Q](https://www.youtube.com/watch?v=KWrzdJY4G9Q)

**Test Yourself**

The normal pacemaker of the heart is the:

1. AV node
2. Bundle of His
3. SA node - Correct

**Section 4: Electrode Placement and Lead Selection**

**Overview**

There are many types of cardiac monitoring systems, but they all generally consist of a monitor
screen on which the ECG is displayed, along with a printer for rhythm strip interpretation and documentation. Some monitoring systems are stationary where the leads and cable are mounted into the fixed monitor. This type of system requires the patient to be confined to a bed or chair. Other monitoring systems are portable with the transmission of the ECG signal through a telemetry or antennae system, thereby allowing the patient to be ambulatory.

Each monitoring system can be set with customized parameters (e.g. the monitor will automatically print a strip if the heart rate drops below 50 or is above 120 beats/min). Some work areas have only one lead monitoring choice, while others are able to monitor in two or more simultaneous leads. Please become familiar with the equipment in your work setting.

Section 4: Electrode Placement and Lead Selection

Electrodes

The ECG records the electrical activity of the heart by using skin sensors called electrodes. Electrodes are adhesive pads that contain a conductive gel and are attached to the patient’s skin.

Having both good skin contact and adequate conductive jelly in the center of the electrode will help assure a good ECG signal is received. The ECG electrode conductive jelly typically dries up within two to three days. When in doubt, replace all electrodes and write the date on the topside of the electrode.

Additional tips for good electrode to skin connections include:

- Remove hair prior to applying the electrode.
- Clean perspiration and any bodily fluids from skin.
- Use a dry towel to mildly “roughen up” or abrade the skin before electrode application (Jones, 2008).

Section 4: Electrode Placement and Lead Selection

Leads

A lead is a record of electrical activity between two electrodes. There are three types of leads: standard limb leads, augmented leads, and precordial leads. This module will focus on the standard limb leads and will not explore the 12 lead ECG which makes use of the augmented and precordial leads.

Each lead has a positive (+) and a negative (-) electrode. When you move the “lead selector” on the ECG machine, you are able to change the polarity of electrodes and thus obtain different lead selections without actually moving the electrodes or lead cables.

Section 4: Electrode Placement and Lead Selection

ECG Deflections: Isoelectric, Upright, Negative, and Biphasic

Isoelectric Line

Each waveform produced is related to a specific electrical event within the heart. When electrical activity is not detected, a straight line is recorded. This is called the electrical baseline or isoelectric line. Although this only occurs in short intervals within the cardiac cycle, we do see an isoelectric line on the ECG when the heart is polarized and awaiting its next contraction.
Section 4: Electrode Placement and Lead Selection

Upright Deflections
A basic rule of electrocardiology refers to the flow of electricity through the heart and out to the skin electrodes. The rule states that if the electricity flows toward the positive electrode, the patterns produced on the graph paper will be upright.

Downward Deflections
Based on the above principle, when the flow of electricity through the heart is away from a positive electrode, it produces a negative deflection on the ECG (Jacobsen, et al., 2014).

Biphasic Deflections
When some of the electricity or conduction current is traveling toward and some travels away from the positive electrode, the ECG recording will have a QRS complex, which are both up and down. This is called a biphasic waveform.
The placement of electrodes for monitoring the ECG allows you to see a single view of the electrical pattern of the heart. Monitoring systems typically have either a 3-lead or a 5-lead system. The 5-lead system has a standardized electrode set-up (electrodes and leads are placed according to the visual illustration provided by the manufacturer). The five lead systems have the benefit of allowing the healthcare provider to monitor in two or more concurrent leads. The 3-lead system allows for monitoring in only one lead at a time. Electrode and lead placement for the 3-cable system is described on the following page (Jones, 2008).

Section 4: Electrode Placement and Lead Selection

Lead I

In Lead I, the negative electrode is under the right clavicle. The positive electrode is under the left clavicle. When the heart depolarizes, the + electrode senses electrical movement coming toward it. Lead I is therefore an upright tracing.

Lead II

In Lead II, the positive electrode is below the left pectoral muscle and the negative is below the right clavicle. Since the left side of the heart has a larger muscle mass (remember the left ventricle is much thicker than the right ventricle), the positive electrode in Lead II senses the current traveling toward the electrode. Lead II is an upright tracing and is therefore a popular lead for monitoring by most healthcare providers. Due to the large upright deflection and ease in "synchronizing" with the "R" wave, Lead II is also the recommended lead of choice for electrical cardioversion.

Section 4: Electrode Placement and Lead Selection

Nursing Tip: With a 3-Lead monitoring system, the ECG cables are often color-coded for ease of application and to reduce confusion about electrode to lead location. The negative lead is usually white, the positive lead is red, and the ground lead is black, green, or brown. For a Lead II set up, a popular phrase is: “white-to-right, red-to-ribs, and black left over.” Another phrase is “smoke over fire.” This little phrase reminds us that the black lead should be on the upper chest (i.e. smoke), while the red lead is on the same side but on the lower chest near the rib area (i.e. “fire” is the red lead).
Lead III
Lead III is displayed by attaching the positive electrode beneath the left pectoral muscle and the negative below the left clavicle. Again, the positive electrode senses electrical current traveling towards it, thereby recording an upright waveform or deflection.

MCL 1 or Modified V1
Another popular monitoring lead is the MCL1 lead (or connect this lead, the negative electrode is placed near the usually under the outer third of the left clavicle, and the electrode is placed to the right of the sternum in the fourth space.

Section 4: Electrode Placement and Lead Selection
5 Lead Set Up
The placement of 3 electrodes for monitoring the ECG allows you to see a single view of the heart’s electrical pattern. 5-lead monitoring systems provide more than one view. The 5-lead system has a standardized electrode setup (electrodes and leads are placed according to the visual illustration provided by the manufacturer). The five lead systems have the benefit of allowing the healthcare provider to monitor in two or more concurrent leads. (Note: The 3-lead system allows for monitoring in only one lead at a time).

Nursing Tip: With a 5 Lead System, the two upper electrodes are placed below the right and left clavicles on the upper chest. The two lower electrodes are placed below the right and left ribs. Precise placement of these electrodes is not required; placement in these general areas will be fine. The “C” or “chest” electrode, however, is different. The “C” electrode (often times brown in color) must be precisely placed at the 4th intercostal space to the right of the sternum. Many healthcare providers are not familiar with the importance of finding this anatomical location. Correct electrode placements for the “C” electrode will ensure an accurate recording of V1. Remember the V1 recording is used to assist with differentiation of Supraventricular Tachycardia, vs. Ventricular Tachycardia, or Bundle Branch Blocks and the continued assessment of potential anterior wall ischemia & injury in patients with chest pain or myocardial infarction.

Test Yourself
Which lead is recommended for electrical cardioversion?
1. Lead I
2. Lead II - Correct
3. Lead III
Section 5: The ECG Paper
The ECG paper is graph paper that is made up of small and larger, heavy-lined squares. The smallest squares are one millimeter wide and one millimeter high. There are five small squares between the heavier lines.

The ECG paper comes out of the printer at constant and standardized speed. On the ECG graph paper, time is measured in seconds along the horizontal axis. Each small square is 1 mm in length and represents 0.04 seconds. This is something that you will need to memorize. Each larger square is 5 mm in length and therefore represents 0.20 seconds.

Voltage or amplitude is measured along the vertical axis. The size or amplitude of a waveform is measured in millivolts or millimeters. The ECG machine is sensitive and may require periodic calibration. One small square on the vertical axis is equal to 1 millimeter (mm). When properly calibrated, a one millivolt electrical signal will produce a deflection measuring exactly 10 mm in height.

Section 5: The ECG Paper
The diagram below illustrates the configuration of ECG graph paper and where to measure the components of the ECG waveform:

ECG Graph Paper and the ECG Wave Form Components

To evaluate an ECG rhythm strip, it is standard practice to print a strip of at least a six second duration. This type of ECG print out will be adequate for the majority of rhythm interpretations. A continuous strip can always be printed especially when unusual rhythms present and require a closer inspection (Jones, 2008).

Test Yourself
On ECG graph paper, a large square represents:
0.04 seconds
0.10 seconds
0.20 seconds - Correct

Section 6: ECG Waveforms
The ECG is a recording of the electrical impulses produced by the heart. The term arrhythmia literally means the “absence of” or “without a rhythm.” The term dysrhythmia is used when there is abnormal cardiac electrical impulses and conduction. Healthcare professionals use both terms interchangeably. Do not let this confuse you.

The body acts as a giant conductor of electrical currents. As you remember, any two points on the
body may be connected by electrical leads (electrodes) to register an ECG or to monitor the rhythm of the heart. The tracing recorded from the electrical activity of the heart forms a series of waves and complexes that have been arbitrarily labeled (in alphabetical order) the P, Q, R, S, and T waves. These waves or deflections occur in regularly occurring intervals in the healthy individual (Heitman, et al., 2012; Jacobson, et al., 2014).

**Normal ECG Waveforms and Intervals**

![ECG Waveforms and Intervals](image)

### Section 6: ECG Waveforms

#### P Wave

Electrical impulses originating from the SA node are represented on the ECG with a waveform called a **P wave**. The P wave is generated after the SA node fires and depolarizes the right and left atria. The beginning of the P wave is recognized as the first upward deflection from the baseline. It resembles a small upward “hill” or “bump” and once completed, returns to the ECG baseline. Locate the P wave in the Normal ECG Waveforms and Intervals figure displayed.
Section 6: ECG Waveforms

PR Interval

When the impulse leaves the atria and travels to the AV node, it encounters a slight delay. The tissues of the node do not conduct impulses as fast as the other cardiac electrical tissues. This means that the wave of depolarization will take a longer time to get through the AV node. On the ECG, this is represented by a short period of electrical inactivity called the **PR interval** (PRI).

The PR interval extends from the beginning of the P wave (the beginning of atrial depolarization) to the onset of the QRS complex (the beginning of ventricular depolarization). It should not exceed 0.20 seconds as measured on ECG graph paper, where each small square represents 0.04 seconds. In other words, the PR interval should not exceed five little boxes in width. This is an important interval to memorize (Heitman, et al., 2012; Jacobson, et al., 2014).

Changes in conduction through the AV node are the most common cause of changes in the PR interval. The P to R interval is important in identification of heart blocks. We will cover this topic later.

QRS Complex

The ventricular depolarization is shown on the ECG by a large complex of three waves: the Q, the R, and the S waves. Together, these three waves are called the **QRS complex**. The QRS complex represents the electrical depolarization of the ventricles. Identify the QRS complex in the QRS Complex figure. Note how the QRS voltage or amplitude is much higher than the height of the P wave. This is because ventricular depolarization involves a greater muscle mass and creates a larger complex.

Following the P wave, the Q wave is the first negative, or downward deflection. The R wave is the first positive or upward deflection following the P wave. The negative wave following the R wave is known as the S wave.

Each QRS complex can look a bit different. In fact, some QRS complexes are lacking a Q wave or others may lack the S wave. Regardless of the appearance, they are always generically called the “QRS” and still indicate depolarization of the ventricles (Heitman, et al., 2012; Jacobson, et al., 2014).
QRS Complex

Section 6: ECG Waveforms
Several different configuration of the QRS complex are shown in the figure below. Can you identify the Q, R, and S components in the figure below?

The upper limit of normal duration of the QRS complex is less than 0.12 seconds or three small boxes. Place one leg of your caliper on the beginning of the Q wave and place the other leg of the caliper on the S wave where it meets the ST segment. A wide QRS complex (more than 0.12 seconds) may signify delayed conduction in one or more of the bundle branches.

The QRS complex reflects depolarization of the ________.
Answer: ventricles
Section 6: ECG Waveforms
ST Segment
The ST segment begins at the end of the S complex and ends with the onset of the T wave. The ST segment represents the early part of repolarization of the ventricles. The ST segment normally sits on the baseline or isoelectric line. It is also normal if the ST segment is slightly elevated or below the isoelectric line (no greater than one millimeter in either direction). Greater than 1 mm ST segment elevation or depression can be indicative of myocardial ischemia or injury (Heitman, et al., 2012; Jacobson, et al., 2014).

The T Wave
Ventricular repolarization is represented on the ECG by a T wave. The beginning of the T wave is identified at the point where the slope of the ST segment appears to become abruptly or gradually steeper. The T wave ends when it returns to the isoelectric baseline.

Test Yourself
The P wave represents:
1. Ventricular depolarization
2. Atrial depolarization - Correct
3. Atrial repolarization

Section 6: ECG Waveforms
Summary
The key to rhythm interpretation is analysis of various waveforms and interrelations of the P wave, the PR interval, and the QRS complex. The ECG should be analyzed with respect to its rate, rhythm, site of the dominant pacemaker, and the configuration of waveforms. These skills require repeated practice.

Practice Exercise
Analyze the strip. Look at the P waves. Note the QRS complex is really only a Q and an R wave. The ST segment is below the isoelectric line. The T wave is upright.

- Circle all of the P waves.
- Measure the PR interval. Using your calipers, measure from the beginning of the P wave upstroke to the point where the P wave joins the Q wave. The PR interval is ______ seconds.
- Measure the QRS width. Measure from the beginning of the Q wave down slope to the end of the R wave (remember there is no “S” wave in this tracing, but we still call it a QRS). The QRS width is _________ seconds.
- Circle all T waves. Do the T waves follow the direction of the R waves?
Yes or No?

Practice Exercise Answers

- PR interval is 4 small boxes or .16 seconds (normal is <.20 seconds).
- QRS width is 2-2.5 boxes or .08 -.10 seconds (normal is < .12 seconds).
- Yes, the T waves are upright and follow the direction of the R wave (which is normal).

Section 7: Rate Measurement

The patient’s heart rate reveals a great deal of information. If the rate is slow (under 60 beats per minute), we call it bradycardia. If the heart rate is fast (over 100 beats per minute), then it is called tachycardia.

ECG monitors display the heart rate. When an ECG strip is printed, most printers provide heart rate information at the top of the strip. Never the less, you might be in a situation where you must calculate the heart rate from the ECG recording. There are numerous methods and formulas, which can be used to calculate a heart rate from the ECG.

Section 7: Rate Measurement

The Six Second Method

This method can be used with either regular or irregular rhythms and provides a rough estimate (but not precise) of heart rate. Print a six second strip (see text box if you are not sure how to do this). Count the number of R waves in a six second strip and multiply by 10. For example, if there are seven (7) R waves in a six second strip, the heart rate is approximately 70 or (7x10=70). Let’s give it a try. Look at the rhythm below and count all the R waves.

What is the approximate heart rate of the ventricles?

![ECG Strip]

**Answer:**

*There are 8 R waves, so our patient’s rate is 8 x 10 or 80 beats per minute.* Wasn’t that easy?

Six Second ECG Strip

Paper Recording

- Look for markers (or hatch-marks) on the top bar of the ECG paper
- Most markers are automatically printed every 1 or 3 seconds
- Print a six second strip
- Add up the number of R waves and multiply by 10
Section 7: Rate Measurement

Large Box Method

Count the number of large squares between two consecutive R waves. Divide this number into 300 for a ventricular rate. For example, if there are four large squares between regular QRS complexes, the heart rate is 75 \((300/4=75)\).

For an atrial rate, count the number of large boxes between two consecutive P waves and also divide into 300 (Palmer, 2011).

Small Box Method

This method also uses an “R to R” or “P to P” measurement, but is more precise because we use the smaller ECG boxes to help us calculate the heart rate. To calculate the ventricular rate, count the number of small boxes between two consecutive R waves and divide by 1500. To calculate the atrial rate, count the number of small boxes between two consecutive P waves and divide by 1500.

Practice using all three types of calculation methods until you find the one you like best. Remember, if you only have a short rhythm strip (<0.06 seconds), you will need to use either Method #2 or #3 (Palmer, 2011).
Section 8: Format for ECG Interpretation

The ECG tracing provides a variety of clues as to what is happening within the heart. These clues include heart rate, regularity or irregularity of the rhythm, interval measurements and characteristics of each individual waveform. Think of the ECG strip as a unique fingerprint in which you are the detective conducting the investigation. Like a detective, you will need to pay attention to details.

In addition to a detailed analysis, you will also need a “recipe” for ECG interpretation, just like a cook needs a recipe for a complex dessert. If you follow the interpretation “recipe” each time you analyze a strip, your skills will grow and your interpretations will be consistently accurate.

Remember to print a six second strip (or longer) and to use your calipers for measurements each time you begin an interpretation. Follow the six basic steps (your recipe) for rhythm interpretation (Palmer, 2011).

**Six Basic Steps for Rhythm Interpretation**

1. **Rate:** (Calculate the heart rate (HR) or note the HR from the monitor)
2. **Regularity:** (Measure the regularity or rhythm of the R waves)
3. **P-wave Examination:** Is there one P wave before each QRS? (there should be)
4. **P to R Interval:** (Measure the P to R interval - Is it within normal limits? It is consistent?)
5. **QRS Width:** (Measure the duration of the QRS complex)
6. **Rhythm Interpretation**

**Section 8: Format for ECG Interpretation**

**Step 1: Rate**

Calculate both the atrial and ventricular rates. Normally the atrial rate is the same as the ventricular rate. Is this true in the ECG strip you are analyzing? Remember the normal heart rate for most individuals falls between the range of 60-100 beats/minute, with most persons between 60-80 beats/minute.

**Normal Findings:**
- The HR should be between 60-100 beats/minute.
- The atrial rate should be the same as the ventricular rate.

**Abnormal Findings:**
- Heart rates less than 60 beats/minute are typically labeled as slow or bradycardic.
- Heart rates greater than 100 beats/minute are typically labeled fast or tachycardic.

**Section 8: Format for ECG Interpretation**

**Step 2: Regularity (or the Pattern of the Rhythm)**

Assess the regularity of the rhythm.

To assess the regularity, you will need to place the legs of your calipers on two consecutive R waves. This is your “R to R” or “R-R” interval. Without moving the width of the calipers, march through the rhythm as you travel from R wave to R wave. Do the R waves follow a regular pattern? If so, the ventricular rhythm is called **regular rhythm.** Normal ECG rhythms are regular in their pattern (Palmer, 2011).
Section 8 Format for ECG Interpretation

Step 2: Irregular Rhythm

If the R-R interval varies in the number of ECG small boxes between them, you are dealing with an irregular rhythm. Do the same type of assessment with the atrial rhythm. Put your calipers at the beginning (or upslope) of a P wave. Put the other end of your caliper at the beginning of the next P wave. This is the P-P interval. Lift your calipers and begin marching through the strip looking for the pattern of regularity of the P waves. If the SA node is firing at a constant beat, the P-P interval will be regular.

Section 8: Format for ECG Interpretation

Normal Findings:
- The R-R intervals are regular
- The P-P intervals are regular
- There is one P for every QRS

Abnormal Findings:
- The R-R intervals are irregular
- The P-P intervals are irregular
- There is more than one P for each QRS
Very few rhythms are irregular. For example, atrial fibrillation is always irregular (more on this rhythm later). Therefore, if your rhythm is regular it cannot be atrial fibrillation. Look at the strips below. Note that the R-R intervals are **regular** in the first tracing. The second tracing has an **irregular** pattern with the R-R intervals. This is a helpful clue in your interpretation process (Palmer, 2011).

**Nursing Tip:** Many healthy individuals have heart rates below 60 beats/minute, especially athletes. Always check the patient’s blood pressure to assess the hemodynamic response to a slow or fast heart rate, especially when there is a rise or fall of greater than 20 beats/minute.

### Section 8: Format for ECG Interpretation

#### Step 3: P Wave Examination

Step 3 is the examination of the P wave. First, you must go on a “P hunt” and find the P waves. Once you have identified them, assess their characteristics.

**Normal Findings**
- P waves should be regular (march out the P-P intervals with your calipers)
- P waves have a symmetrical shape, usually upright and rounded
- P waves should all look alike (uniform) and should point in the same direction
- There should be one P for every QRS (or a 1:1 relationship)

**Abnormal Findings**
- A P wave is not followed by a QRS complex
- There are more P waves than QRS complexes

**Nursing Tip:** Remember that the P wave represents atrial depolarization.

#### Step 4: P to R Interval

Remember that the P to R interval represents the time it takes an impulse to travel from the atria through the AV node. The P to R interval is measured from the beginning of the P wave to the beginning of the QRS complex. This is a bit confusing as you might think it is a measurement from the beginning of the P wave to the beginning of the R wave... but it is actually only measured from the beginning P to the beginning of the Q wave. Think of it as a “P to Q measurement” despite the fact that it is called a PR interval (Palmer, 2011).
Section 8: Format for ECG Interpretation
Step 4: P to R Interval

Normal Findings:
- The PR interval (or time travel from SA to AV nodes) is between 0.12 to 0.20 seconds.
- The PR intervals are constant throughout the rhythm.

Abnormal Findings:
- The PR interval is > 0.20 seconds (this might indicate delayed travel time from SA to AV node).
- The PR interval is irregular in measurement (irregular or varying PR intervals may indicate some type of SA-AV conduction problem and possible conduction heart block) (Palmer, 2011).

Nursing Tip: Normal PR interval is 0.12-0.20 seconds (3-5 small boxes).

Section 8: Format for ECG Interpretation
Step 5: QRS Complex

The QRS complex represents ventricular depolarization. The QRS complex consists of three waves: the Q wave, the R wave, and the S wave. It is measured from the beginning of the Q wave to the end of S wave. Normal ventricular conduction and depolarization takes no more than 0.12 seconds (Palmer, 2011).

Normal Findings:
- All the QRS complexes have uniformity throughout (the same size, shape and direction).
- All QRS complexes are of equal duration or width.
- The R to R interval between each QRS is regular.

Abnormal Findings:
- The QRS complexes vary in shape, width and direction.
- The QRS complex is >0.12 seconds wide.
- The R to R interval between each QRS is irregular.

Section 8: Format for ECG Interpretation
ST – T Wave

The ST segment and T wave represent ventricular repolarization. The cells are returning back to their polarized status and the heart is getting ready for yet another contraction.

Normal Findings:
- The ST segment should be electrically neutral (or near neutral) and should be sitting on the isoelectric baseline (no greater than 1 mm above or below the isoelectric line is normal).
- The T wave is upright whenever the R wave is upright. The T wave deflects downward when the R wave is downward.

Abnormal Findings:
- There is > 1mm ST segment elevation or depression from the isoelectric line.
- The T wave is in the opposite direction than the R wave.
Bundle Branch Blocks

Do you hear this term a lot, but are not quite sure what it is all about? Bundle branch blocks are outside the scope of this course. Since there is such a high level of interest in them, and some ECG learners may be ready to tackle this concept, a short description of bundle branch blocks is included in Appendix I. Bundle branch blocks will not be included on the post test.

Section 8: Format for ECG Interpretation
Step 6: Rhythm Interpretation

Believe it or not, after completing Step 5, you are ready to make an educated decision on naming the correct rhythm. Remember to correlate information obtained in Steps 1-5 along with your understanding of the heart’s electrophysiology. Rather than pure memorization, if you can integrate the electrophysiology with the rhythm interpretation your patient care priorities and potential treatments will make a lot more sense.

Nursing Priorities and Potential Treatments

Interpreting the actual ECG rhythm is only the beginning of the assessment and care for your patient. You cannot be successful in your practice if you only know how to interpret ECG. As healthcare providers, you must be able to respond with appropriate priorities and understand initial treatments. Comprehensive treatments for each type of ECG rhythm are beyond the scope of this module, however, initial or “first steps” will be discussed.

Section 8: Format for ECG Interpretation

Look at the following 6-second rhythm strip and begin to answer the questions below:

1. Heart rate: __________ Normal/Fast/Slow
2. Regularity of the rhythm:
   • Are the P to P intervals regular? Yes/No
   • Are the R to R intervals regular? Yes/No
3. P waves:
   • Are P waves present? Yes/No
   • Is there one P proceeding each QRS? Yes/No
4. PR interval: __________ <.20 seconds (normal) or > .20 seconds
5. QRS width: __________ <.12 seconds (normal) or >.12 seconds
6. Rhythm interpretation: __________________________________________
Practice Exercise Answers

1. Heart rate: **Approximately 80 beats/min** (8 R waves in a six second strip x 10). Normal.
2. Regularity of the rhythm: **The P to P intervals are regular. The R to R intervals are regular.**
3. P waves: **The P waves are present. There is one P preceding each QRS.**
4. PR interval: 4 sm boxes x .04 sec = **.16 seconds <.20 seconds (normal)**
5. QRS width: 2 sm boxes x .04 sec = **.08 seconds <.12 seconds (normal)**
6. Rhythm interpretation: We are not ready for this yet, but a sneak preview will indicate this is a **Normal Sinus Rhythm.** (More on this soon!!)

Test Yourself

A PR interval of >0.20 seconds is considered a normal finding.

1. True
2. False - Correct

Section 9: The Sinus Rhythms

**Normal Sinus Rhythm**

![ECG Image]

Description

**Normal sinus rhythm** (NSR) is also simply called **sinus rhythm** (both terms are used interchangeably). NSR is the result of the dominant pacer function from the SA node. In addition to the healthy SA nodal function, all of the conduction pathways are working normally (the AV node, junction, bundle of His, right and left bundles and the Purkinje fibers). The sinus node is firing at a regular rate of 60-100 times per minute. Each beat is conducted normally through to the ventricles.

**ECG Criteria:**

1. **Heart rate:** 60 – 100 bpm
2. **Rhythm:** Atrial regular. Ventricular regular
3. **P waves:** Upright and uniform (all of the P waves look alike). One P precedes every QRS
4. **PR interval:** 0.12 - 0.20 seconds (less than one big box on the graph paper)
5. **QRS width:** ≤ 0.12 seconds

**Nursing Priorities:** There are no nursing priorities.

**Potential Treatments:** There are no potential treatments.
Important Self-Assessment Note

NSR is the standard against which all other rhythms are compared.

If you are not clear on this rhythm, do not advance in this module. To have your questions answered and to clear up any confusion, please re-read the previous sections and consult with a colleague BEFORE continuing with this module.

Section 9: Sinus Bradycardia

Description

Sinus bradycardia (SB) is characterized by a decrease in the rate of atrial depolarization due to slowing of the SA node. The sinus node is the pacemaker, firing regularly at a rate of less than 60 times a minute. Each impulse is conducted normally through to the ventricles.

Possible Causes:

- Increased vagal (parasympathetic) tone (vomiting, carotid sinus massage)
- Hyperkalemia
- Increased intracranial pressures
- Possible result of inferior myocardial infarction (MI)
- Side effects from beta-adrenergic blockers, sympatholytic drugs, digoxin, or morphine
- Normal effect, especially in athletes (Jacobsen, et al., 2014)

ECG Criteria:

1. Heart rate: Less than 60 bpm.
3. P waves: Upright and uniform. One P precedes every QRS.
4. PR interval: 0.12 -0.20 seconds.
5. QRS width: ≤ 0.12 seconds.

Nursing Priorities:

- Check your patient’s blood pressure, assess for syncope, and shortness of breath (SOB).
- You patient may need to lie down to prevent potential falls.
- Keep in mind that it may be normal for some individuals to have sinus bradycardia (e.g. athletes).

Potential Treatments:

- Asymptomatic: Observation.
Section 9: Sinus Tachycardia

Description
Sinus tachycardia (also called Sinus Tach) is characterized by a rapid (> 100 bpm) rate of discharge of the SA node. The sinus node is discharging at a rate > 100 and the remainder of the conduction follows the normal pathway.

Possible Causes:
- Normal cardiac response to demands for increased oxygen need during pain, fever, stress, dehydration and exercise.
- Caffeine, nicotine ingestion.
- Hyperthyroidism.
- Post MI; early sign of heart failure (Jacobsen, et al., 2014).

ECG Criteria:
1. **Heart rate**: ≥ 100 bpm to 160 bpm.
2. **Rhythm**: Regular.
3. **P waves**: Upright and normal. One P precedes every QRS.
4. **PR interval**: 0.12 - 0.20 seconds.
5. **QRS width**: ≤ 0.12 seconds.

Nursing Priorities:
- Check your patient’s blood pressure, assess for syncope, palpitations, or SOB.
- Your patient may need to lie down to prevent potential falls.
- Patient may have lower blood pressure due to decreased diastolic ventricular filling time associated with the tachycardia.

Potential Treatments:
- Asymptomatic: Observation.
- Symptomatic: Treat the underlying cause (dehydration, anxiety, etc). Drugs such as beta blockers may be given to slow the HR (Jacobsen, et al., 2014).

Test Yourself
The interpretation of the rhythm displayed is:
1. Normal sinus rhythm
2. Sinus bradycardia - Correct
3. Sinus tachycardia
Section 10: Atrial Dysrhythmias PACs
Premature Atrial Contractions (PACs)

As previously mentioned, the intrinsic pacemaker of the heart is the sinus (SA) node, and normally it initiates each beat. Pacemaker stimuli can also arise from other parts of the heart – the atria, the AV junction, or the ventricles. The terms “ectopy” or “ectopic beat” are used to describe these beats that do not emanate from the SA node. Ectopic beats are often premature; that is, they come in early or before the next sinus beat is due.

Description
A premature atrial contraction (PAC) results from an ectopic stimulus that arises from somewhere in either the left or the right atrium, but not in the sinus node. The atria are depolarized from the ectopic stimulus, but the remainder of the conduction is typically normal through the AV Node-Junction and downward into the bundle branches (i.e. normal PR and QRS morphology and intervals).

Possible Causes:
PACs are very common and may occur in persons with a normal heart or in persons with virtually any type of organic heart disease. PACs do not imply that a person has cardiac disease and may be seen with caffeine intake and with emotional stress.

Other causes include:
- Administration of sympathomimetic agents (epinephrine, theophylline).
- Electrolyte abnormalities.
- Myocardial ischemia or injury.
- Digoxin toxicity.

ECG Criteria:
1. **Heart rate**: Typically normal.
2. **Rhythm**: Underlying rhythm is typically regular with early premature beats.
3. **P waves**: Atrial depolarization is premature, occurring before the next normal P wave. Since the impulse originates outside the SA node, the P wave may have a different shape - often notched, peaked or buried in the proceeding T wave.
4. **PR interval**: Maybe normal, shorter or longer than normal PR interval, depending on origin of the PAC.
5. **QRS width**: Typically normal but may be prolonged if the PAC is aberrantly conducted through the ventricles.

Section 10: Atrial Dysrhythmias PACs
Other Types of PACs
There are a few variations from the above description. On occasion, a PAC may not conduct into the ventricles or the PAC may conduct into the ventricles in an abnormal way.

Nonconducted PACs: If the PAC occurs very prematurely (or close to the preceding T wave), the early atrial depolarization might be too early for the right and left bundles to conduct the impulse. This
type of PAC cannot be conducted down into the ventricles. In this situation, look for an early P wave (which might also be buried in the preceding T wave). The early PAC does not conduct into the ventricles, thus there is no QRS for this one cardiac cycle.

**Aberrantly conducted PACs:** If the impulse should happen to travel abnormally through the ventricles, the QRS may be prolonged. This can happen if either the right or left bundle branches are not ready to depolarize and result in a temporary block. If the QRS is wide following a PAC, it will be called an aberrantly conducted PAC.

**Nursing Priorities:**
- Intervention not typically required.
- Heart rate may be irregular during episodes of PACs so assess the pulse for one full minute.

**Potential Treatments:**

**Asymptomatic**
- Observation and ECG monitoring for frequency and trends.
- Explore potential underlying causes (caffeine intake, stress reduction, myocardial abnormalities, etc).

**Symptomatic**
- Treatment is typically centered around observation and monitoring for increased frequency.
- Be aware that individuals may complain of palpitations or feeling a “skipped” heart beat with an irregular pulse. Explain to them the reason for these feelings.
- Increased number of PACs may be a forerunner of the development of atrial fibrillation or other atrial dysrhythmias (Jacobsen, et al., 2014).

**Section 10: Atrial Flutter**

![ECG Waveform](image)

**Description**

**Atrial Flutter** is a dysrhythmia that is the result of a flawed reentry circuit within the atria. It is often described as resembling a sawtooth or picket fence. These *flutter waves* should not be confused for P waves.

The AV node is a wonderful protective mechanism. Imagine the atria depolarizing at a rate of 250 to 350 bpm. If all of these atrial depolarizations were conducted down into the ventricle, the patient’s ventricles would likely begin to fibrillate.

Think of the AV node as the central train station where numerous train tracks merge. The central station only lets some of the trains through to avoid congestion. The AV node helps to protect the ventricles by only allowing some of the atrial depolarizations to conduct down through the bundle of His into the bundle branches and on to the ventricles.
When the ventricular rate is < 100 bpm, we call this “controlled atrial flutter.” If the ventricular rate is > 100 bpm, it is labeled “uncontrolled atrial flutter.” Since the ventricles always have more time to fill during diastole when the HR is under 100, our goal is to have controlled atrial flutter. This can often be accomplished with drug therapy.

In the setting of atrial flutter, coordinated contraction of the atria is absent. The patient has therefore lost their atrial kick with potential loss of cardiac output and lower blood pressure.

Possible Causes:
- Acute or chronic cardiac disorder, mitral or tricuspid valve disorder, cor pulmonale, pericarditis.
- Post MI complication (usually transient).
- Hyperthyroidism.
- Alcoholism.
- Post cardiac surgery (usually transient) (Jacobsen, et al., 2014).

ECG Criteria:
1. Heart rate: Atrial rate is 250-350 bpm. Ventricular rate varies according to AV node conduction.
2. Rhythm: Atrial regular; ventricular may be regular or irregular (again, depending on AV node conduction).
3. P waves: Absent. Only flutter or sawtooth looking waveforms.
4. PR interval: Not applicable.
5. QRS width: ≤ 0.12 seconds.

Nursing Priorities:
- Check your patient’s blood pressure, assess for syncope, palpitations, or SOB.
- Your patient may need to lie down to prevent potential falls.
- Patient may have lower blood pressure due to loss of atrial kick.

Potential Treatments:
- Asymptomatic: Observation and rate control.
- Normal heart function: Control ventricular rate by administering beta blockers (esmolol) or calcium channel blockers (diltiazem or verapamil).
- Impaired heart function: Control ventricular rate by administering digoxin, diltiazem, or amiodarone.
- Symptomatic: If hemodynamically unstable, synchronized cardioversion at 100-200 monophasic joules (Jacobsen, et al., 2014).
Section 10: Atrial Fibrillation

Description
Atrial fibrillation (often called “a-fib” or “atrial fib”) may result from multiple areas of re-entry within the atria or from multiple ectopic foci. The atrial electrical activity is very rapid (approximately 400 bpm), but each electrical impulse results in the depolarization of only a small islet of atrial myocardium rather than the whole atrium. As a result, there is no contraction of the atria as a whole. Since there is no uniform atrial depolarization, there is no P wave. The chaotic electrical activity does produce a deflection on the ECG, referred to as a fibrillatory wave.

Fibrillatory waves vary in size and shape and are irregular in rhythm. Fibrillatory waves look different from the sawtooth waves of atrial flutter. Transmission of these multiple atrial impulses into the AV node is thought to occur at random, resulting in an irregular rhythm. Some impulses are conducted into but not through the AV node (they are blocked within the AV node). Remember that the ventricular rhythm is always irregular in atrial fibrillation.

When the ventricular rate is < 100 bpm, we call this “controlled atrial fibrillation.” If the ventricular rate is > 100 bpm, it is labeled “uncontrolled atrial fibrillation.” Since diastolic filling is enhanced when the HR is under 100, our goal is to have controlled atrial fibrillation. This can often be accomplished with drug therapy (Jacobsen et al., 2014).

Possible Causes:
- Mitral valve disorders.
- Rheumatic heart disease, MI, hypertension, coronary artery disease (CAD), heart failure, pericarditis.
- Chronic obstructive pulmonary disease (COPD).
- Digoxin toxicity.
- Post cardiac surgery (usually transient).

ECG Criteria:
1. **Heart rate**: Atrial rate 350-400 bpm. Ventricular rate is variable.
2. **Rhythm**: Ventricular rate is irregular (one of the hallmark signs of atrial fibrillation).
3. **P waves**: Absent. Only atrial fibrillatory waves (or small looking bumps) are seen.
4. **PR interval**: Not applicable.
5. **QRS width**: ≤ 0.12 seconds.

Nursing Priorities:
- Check your patient’s blood pressure, assess for syncope or SOB.
- Palpitations are commonly felt as a result of the irregular, and often rapid, heart rate.
- Your patient may have lower blood pressure due to loss of atrial kick.
Potential Treatments:
- **Asymptomatic**: Observation and rate control.
- **Normal heart function**: Control ventricular rate by administering beta blockers (esmolol) or calcium channel blockers (diltiazem or verapamil).
- **Impaired heart function**: Control ventricular rate by administering digoxin, diltiazem or amiodarone.
- **Symptomatic**: If hemodynamically unstable, synchronized cardioversion (Jacobsen et al., 2014).

**Nursing Tip:**
Before electrical cardioversion is attempted, consider anticoagulation to prevent embolic complications.

Test Yourself
**Interpretation of the rhythm displayed is:**
1. Premature atrial contraction
2. Atrial fibrillation
3. Atrial flutter - Correct

Section 11: Junctional Escape Rhythm

As you probably remember, the AV node is a group of specialized cells and its main function is to delay impulses coming from the atria to ventricles, thereby allowing the atria more time to completely contract. Between the AV node and the right and left bundle branches lies the Bundle of His. The area around the Bundle of His is also called the AV junction (where the AV node and the bundles junction together).

**Description**
This AV junction can function as a pacemaker. It initiates impulses at a rate of 40 to 60 beats per minute. Under normal circumstances, the sinus node pacemaker is faster and predominates. If the AV node is not depolarized by the arrival of a sinus impulse within approximately 1.0 to 1.5 seconds, it will initiate an impulse of its own from this junctional area. This is called a **junctional escape complex**.

Junctional escape complex occurs because of failure of the sinus node to initiate an appropriately timed impulse or because of a conduction problem between the sinus node and the AV junction. A repeated series of such impulses is referred to as a **junctional escape rhythm**.

**Unusual or Absent P waves:**
If the AV junction paces the heart, the atria may or may not be stimulated. The electrical impulse must travel in a backward (retrograde) direction to activate the atria. In Leads II, III, and aVF, the P wave will be negative (inverted) if the atria are stimulated. Depending on how the atria responds to the junctional pacemaker, the patient could have:
- No P waves
- An inverted P wave (upside down just preceding the QRS)
- An inverted P wave after the QRS (if atrial depolarization occurs after the QRS)

**Possible Causes:**
- Post MI (damage to SA node).
- Digoxin toxicity (Jacobsen et al., 2014).

**ECG Criteria:**
1. **Heart rate:** 40-60 bpm.
2. **Rhythm:** Ventricular rhythm is regular.
3. **P waves:** May be absent or may occur before, during or after the QRS (due to retrograde conduction).
4. **PR interval:** None (impulses are originating from the AV junction, not the SA node).
5. **QRS width:** ≤ 0.12 seconds (the impulse is traveling down the normal pathways of the right and left bundles).

**Nursing Priorities:**
- Check your patient’s blood pressure, assess for syncope, palpitations, or SOB.
- Lower blood pressure may result from loss of atrial kick and bradycardic heart rate.

**Potential Treatments:**
- **Asymptomatic:** Observation.
- **Symptomatic:** Atropine, dopamine, pacemaker (Jacobsen et al., 2014).

**Section 11: Accelerated Junctional Rhythm/Junctional Tachycardia**

Can the AV junctional area ever “speed up” and pace at a rate faster than 40-60 beats per minute? The answer is “yes, it can.” For all of the same reasons a person might experience a junction escape rhythm, a person might also experience enhanced automaticity of the AV junction area. The result is a junctional rhythm, which depolarizes at a rate of 60-100 bpm. This is an **accelerated junctional rhythm**. If the rate is greater than 100 bpm the rhythm is called **junctional tachycardia** (see above).

It might be tempting to call the ECG tracing a normal sinus rhythm, but make note that uniform looking P waves are absent along with a constant PR interval. The SA node is not working and the junction has taken over as the pacemaker, only a bit faster than its normal intrinsic rate of 40-60 bpm.
Section 11: Accelerated Junctional Tachycardia

Possible Causes of Accelerated Junctional Rhythm:
- Digoxin toxicity (most common cause).
- Hypoxia.
- Cardiomyopathy.
- MI.
- Valve replacement surgery (Jacobsen et al., 2014).

ECG Criteria of Accelerated Junctional Rhythm:
1. **Heart rate**: 60-100 bpm. (Remember: if heart rate is > 100 it is called junctional tachycardia.)
2. **Rhythm**: Ventricular rhythm is regular.
3. **P waves**: May be absent or may occur before, during or after the QRS (due to retrograde conduction).
4. **PR interval**: None (impulses are originating from the junction, not the SA node).
5. **QRS width**: ≤ 0.12 seconds (the impulse is traveling down the normal pathways of the right and left bundles).

Nursing Priorities:
- Check your patient’s blood pressure, assess for syncope, palpitations, or SOB.
- Lower blood pressure may result from loss of atrial kick.

Potential Treatments:
- **Asymptomatic**: Observation.
- **Symptomatic**: Discontinue digoxin therapy. Check potassium levels. If heart rate becomes faster (100-180 bpm) the rhythm is called junctional tachycardia. Now consider treatment with IV amiodarone, beta-adrenergic blockers (esmolol, labetalol, metoprolol), or calcium channel blockers (Jacobsen et al., 2014).

Test Yourself
A common cause of junctional rhythms is:
1. Dopamine overdose.
2. Digoxin toxicity. - Correct
3. Prolonged atrial tachycardia.

Intermission

TAKE A BREAK!!!!

If you have been working at this course without a break, we suggest you take one now. Let some of the information “simmer” in your head. Come back fresh and ready to tackle even more information about interpreting ECGs!
Section 12: Atrioventricular Blocks

Atrioventricular blocks (otherwise known as AV blocks or heart blocks) can be divided into three degrees. First-degree AV blocks are characterized by P to R intervals longer than 0.20 seconds. Second-degree AV blocks are characterized by some P waves being blocked at the AV node. This results in some P waves occurring without following QRS complexes. Third-degree AV block is characterized by a complete dissociation between P waves and QRS complexes.

First Degree AV Block

Description
First-degree AV block is simply a delay in passage of the impulse from atria to ventricles. Unlike its name (which can be confusing), first-degree AV block is not an actual “block,” but rather a delay in conduction. This conduction delay usually occurs at the level of the AV node.

Remember that in normal sinus rhythm, the time it takes the SA node to fire, depolarize the atria and transmit to the AV node is < 0.20 seconds. In first degree AV block the patient has a PR interval of > 0.20 seconds. If the patient’s underlying rhythm is sinus bradycardia, but the PR interval is 0.24 seconds, the interpretation would be “sinus bradycardia with a first-degree AV block.”

Section 12: First Degree AV Block

Possible Causes:
- Drug therapy (digoxin, beta-adrenergic blockers or calcium channel blockers, or antiarrhythmic drugs such as amiodarone).
- Post MI.
- Chronic degenerative disease of the atrial conduction system (seen with aging).
- Hypo- or hyperkalemia.
- Increased vagal tone (Jacobsen et al., 2014; Maryniak, 2012).

ECG Criteria:
1. **Heart rate**: Varies depending on the underlying rhythm.
2. **Rhythm**: Atrial and ventricular regular.
3. **P waves**: Upright and normal. One P precedes every QRS.
4. **PR interval**: ≥ 0.20 seconds and is constant.
5. **QRS width**: ≤ 0.12 seconds.

Nursing Priorities:
- Observe for lengthening PR intervals or development of more serious heart blocks.

Potential Treatments:
- Treatment for first-degree AV block is usually unnecessary as it is typically asymptomatic.
- Treatment typically aims to correct the underlying cause.
- Consult with physician if PR interval is lengthening. Discuss holding medications which slow A-V conduction (Jacobsen et al., 2014; Maryniak, 2012).
Section 12: Second Degree AV Block Type I

There are two categories of second-degree AV block. One is called **Wenckebach (Type I)** and the other is called **Type II**. In both types, the impulse originates in the sinus node, but is conducted through the AV node in an intermittent fashion. Simply stated, not every P wave will be followed by QRS complex. In second-degree AV blocks, some impulses are conducted and others are not. The cause of the non-conducted P waves is related to intermittent AV nodal block. The difference between the two-second degree blocks is related to the pattern in which the P waves are blocked.

**Description**

**Second-degree AV block-Type I** is unique in that it has three different names, and all three are used interchangeably (just to keep us all on our toes!). Second degree AV Block-Type I is also called **Mobitz I** or it can be referred to as **Wenckebach**. Do not let this confuse you as all three names mean the **SAME** rhythm.

We will refer to Second Degree AV Block Type I as “Wenckebach” for the remainder of this course. Wenckebach is characterized by a **progressive prolongation of the PR interval** (so the key to diagnosing this rhythm is by careful examination of each PR interval). The SA node is healthy and fires on time, thus the P to P intervals are regular. Impulses traveling through the AV node take longer and longer to fully conduct until one impulse is completely blocked. The SA node continues to fire right on time (regular P to P intervals) and the cycle of prolongation of PR intervals continues as the pattern is repeated.

The repetition of this pattern results in “group beating,” (e.g. three conducted sinus beats with progressively lengthening PR intervals and a fourth sinus beat that is NOT followed by a QRS). Beats that are successfully conducted have a normal QRS width. Because QRS complexes are periodically dropped, the ventricular rhythm is irregular.

This block almost always occurs at the level of the AV node (rarely at His bundle or bundle branch level), is typically a transient rhythm, and prognosis is good.

**Possible Causes:**

- Drug therapy (digoxin, beta-adrenergic blockers, calcium channel blockers, amiodarone).
- CAD.
- New MI – seen more commonly with acute inferior wall infarctions.
- Rheumatic fever.
- Increased vagal tone.
- Hyperkalemia.
- Myocarditis (Jacobsen et al., 2014; Maryniak, 2012).
ECG Criteria:
1. **Heart rate**: Atrial regular; ventricular rate is slightly slower. Typically between 60-90 bpm.
3. **P waves**: Upright and normal. Some P’s are not followed by a QRS (more Ps than QRSs).
4. **PR interval**: Progressively longer until one P wave is not followed by a QRS complex. After the blocked beat, the cycle starts again.
5. **QRS width**: ≤ 0.12 seconds.

Nursing Priorities:
- Check the patient’s blood pressure and other patient vital signs (often they are normal).
- Assess the patient for possible causes.

Potential Treatments: (Treatment is not typically required.)
- **Asymptomatic**: Observation and monitoring only. If PR interval is lengthening, consult with physician for holding drugs that can slow AV node conduction.
- **Symptomatic**: On a rare occasion, atropine and/or temporary pacing may be considered.

Section 12: Second Degree AV Block Type II

**Description**

Second degree AV block **Type II** is also referred to as Mobitz II (only two names this time 😊). This form of conduction delay occurs below the level of the AV node, either at the bundle of His (uncommon) or the bundle branches (common). A hallmark of this type of second-degree AV block is that there is a pattern of conducted P waves (**with a constant PR interval**), followed by one or more non-conducted P waves. The PR interval does not lengthen before a dropped beat. Remember that the P waves that are successful in conducting through have a **constant PR interval**. Since the SA node is firing in a regular pattern, the P to P intervals again march through in a regular pattern (P-P is regular). Since not all P waves are conducted into the ventricles, the R to R intervals will be irregular and the ventricular response (HR) may be in the bradycardia range.

When the block occurs at the Bundle of His, the QRS may be narrow since ventricular conduction is not disturbed in beats that are not blocked. If the blockage occurs at the level of the bundle branches, conduction through the ventricles will be slower therefore creating a wider QRS complex (>0.12 seconds).

Mobitz II is associated with a poorer prognosis, and complete heart block may develop. Causes are usually associated with an acute myocardial infarction, severe coronary artery disease or other types of organic lesions in the conduction pathway. The patient’s response to the dysrhythmia is usually related to the ventricular rate.
Possible Causes:
- Age related degenerative changes in the conduction system.
- New MI- seen more commonly with acute anterior wall infarctions.
- Post cardiac surgery or complication arising with cardiac catheterization.
- Note: not typically a result of increased vagal tone or drug effects (Jacobsen et al., 2014; Maryniak, 2012).

ECG Criteria:
1. **Heart rate**: Atrial regular. Ventricular rate is typically \( \frac{1}{4} \) to \( \frac{1}{2} \) the atrial rate (depending on the amount of blockage in conduction).
2. **Rhythm**: Atrial regular (P-P is regular). Ventricular irregular.
3. **P waves**: Upright and normal. Some Ps are not followed by a QRS (more Ps than QRSs).
4. **PR interval**: The PR interval for conducted beats will be constant across the strip.
5. **QRS width**: \( \leq 0.12 \) seconds for conducted beats.

Nursing Priorities:
- Check the patient’s blood pressure.
- Assess the patient’s symptoms.
- **Note**: Mobitz II has the potential to suddenly progress to complete heart block (third degree AV block) or ventricular standstill; have a temporary pacemaker nearby!

Potential Treatments:
- **Asymptomatic**: Observation and monitoring only. Hold drugs that can slow AV node conduction. Notify physician. Obtain supplies for pacing should this become necessary.
- **Symptomatic**: If symptomatic bradycardia is present, temporary pacing is initiated. Administer a dopamine infusion if patient is hypotensive.
- **Note**: Atropine must be used with great caution (if at all) with this rhythm. Atropine will increase the sinus note discharge, but does not improve conduction through the AV node, (the location of this block is lower in the conduction system). Acceleration of the atrial rate may result in a paradoxical slowing of the ventricular rate, thereby decreasing the cardiac output (Jacobsen et al., 2014; Maryniak, 2012).

Section 12: Third Degree AV Block
Third-degree AV block is also called complete heart block. This type of dysrhythmia indicates complete absence of conduction between atria and ventricles (the atria and the ventricles are not communicating with one another). The atrial rate is always equal to or faster than the ventricular rate in complete heart block. The block may occur at the level of the AV node, the bundle of His, or in the bundle branches. As in second-degree AV block, this distinction is not merely academic since pathogenesis, treatment, and prognosis may vary considerably, depending on the anatomic level of block.

When third-degree AV block occurs at the AV node, a junctional escape pacemaker frequently will initiate ventricular depolarization. This is usually a stable pacemaker with a rate of 40 to 60 beats per minute. Since it is located above the bifurcation of the bundle of His, the sequence of ventricular depolarization usually is normal, resulting in a normal QRS. This type of third-degree AV block can result from increased parasympathetic tone associated with inferior infarction, from toxic drug effects (e.g., digoxin, propranolol), or from damage to the AV node.

When third-degree AV block occurs below the junction, it is most often due to a block involving both bundle branches. The only escape mechanism available is in the ventricle distal to the site of block. Such a ventricular escape pacemaker has an intrinsic rate that is slow, less than 20-40 beats per minute. Like any depolarization originating in a ventricle, the QRS complex will be wide. It is not a stable pacemaker, and episodes of ventricular asystole are common.

Remember that the rhythm strip reflects two separate processes that are taking place. The SA node continues to control the atria and typically fires at a rate of 60-80 bpm. Since the atria and the ventricles are not communicating, one of the two remaining back-up intrinsic pacemakers will take over. Either the junction will pace the ventricles (rate 40-60 bpm) or the back-up ventricular pacer will discharge (rate 20-40 bpm).

When there are two separate pacemakers controlling the upper and lower chambers of the heart without regard to each other, the situation is called AV disassociation (this is not a “rhythm” but a “condition” and the umbrella term AV disassociation is often used).

On the ECG, you will see normal P waves marching regularly across the strip. The P-P intervals are regular. You will also see QRS complexes at regular intervals. The unique feature is that the P waves and the QRS complexes will not be “talking to each other.” There is no relationship between the P and the QRS waveforms. The PR interval will be totally inconsistent and you may even see P waves superimposed in the middle of QRS complexes. There will be more P waves than QRS complexes (because the intrinsic rate of the sinus node is faster than either the junctional or ventricular rates).

Possible Causes:
- Drug therapy (digoxin, beta-adrenergic blockers, calcium channel blockers, amiodarone).
- New MI – seen more commonly with acute inferior wall infarctions.
- Increased vagal tone.
- Hyperkalemia.
- Myocarditis or rheumatic heart fever.
- Post cardiac surgery or complication arising with cardiac catheterization (Jacobsen et al., 2014; Maryniak, 2012).

ECG Criteria:
1. **Heart rate**: Atrial rate is normal. Ventricular rate is slower. 40-60 bpm if back-up pacer is from the junction or 20-40 bpm if back-up pacer is from the ventricles.
2. **Rhythm**: P-P is regular; R-R is regular (but the two are independent functions).
3. **P waves**: Upright and normal.
4. **PR interval**: No relationship between the P and the QRS waves. No PR interval.
5. **QRS width**: ≤ 0.12 seconds if controlled by the junction; > 0.12 seconds if paced by the ventricle.

**Nursing Priorities:**
- Check the patient’s blood pressure, assess for syncope, palpitations, or SOB.
- Your patient may need to lie down to prevent syncope and/or potential falls.
- Patient may have lower blood pressure due to low ventricular rate.

**Potential Treatments:**
- **Asymptomatic**: Notify physician. Observation and monitoring only. Hold drugs that can slow AV node conduction. Obtain supplies for pacing should this become necessary.
- **Symptomatic**: Notify physician. If symptomatic bradycardia is present, administer atropine and prepare for temporary pacing. Atropine may be effective if the QRS is narrow (AV node level of block) but has little or no effect on wide QRS (bundle-branch level) third-degree block rhythms. Administer a dopamine infusion if patient is hypotensive (Jacobsen et al., 2014; Maryniak, 2012).

**Test Yourself**
Interpretation of the rhythm displayed is:

1. **First degree AV block**
2. **Second degree AV block, type I (Wenckebach)** - Correct
3. **Third degree AV block**

**Section 13: Ventricular Rhythms**

All of the dysrhythmias which you have learned thus far are classified as *supraventricular* dysrhythmias, because they originate from above the ventricles. When a rhythm originates from above the ventricles (and thereby travels down the normal right and left bundles to the Purkinje fibers) the QRS is 0.12 seconds or less. When rhythms originate in the ventricles, they generally have a QRS > 0.12 seconds because they are coming from an area outside the right and left bundle branches. This is an important concept to remember.
Ventricular rhythms are of great importance and can be very dangerous. Our hearts were designed to conduct and contract from the top down (atria to ventricle). When this mechanism is disrupted, we lose our atrial kick and the heart’s efficiency is greatly reduced.

The first dysrhythmia is not an actual rhythm, but an occasional ectopic (abnormal) beat originating from an irritable cluster of cells somewhere in either the right or left ventricle.

Section 13: Premature Ventricular Contractions

Description
A premature ventricular contraction (PVC) is a depolarization that arises in either ventricle before the next expected sinus beat, and is therefore labeled “premature.” They are generally easy to detect because the QRS is wide and bizarre looking. Since PVCs originate in the ventricle, the normal sequence of ventricular depolarization is altered. For example, instead of the two ventricles depolarizing simultaneously, a PVC will cause the ventricles to depolarize at different times or sequentially. In addition, conduction occurs more slowly through the myocardium than through specialized conduction pathways. This results in a wide (0.12 second or greater) and bizarre-appearing QRS. The sequence of repolarization is also altered, usually resulting in an ST segment and T wave in a direction opposite to the QRS complex. After the PVC occurs, you may find a short pause before the next QRS. This is called a compensatory pause. The compensatory pause may or may not be present (Heitman et al., 2012; Jacobson, et al., 2014).

Unifocal PVCs:
When a PVC originates from a single focus, its morphology or waveform characteristics look the same each time. When a PVC looks the same each time, it is called a unifocal PVC (because it originates from one area). All of the PVCs from a unifocal source are identical in appearance.

The strip below is an example of unifocal PVCs.
Multifocal PVCs:
In cases of greater irritability, several ventricular foci might begin to initiate ectopic beats. **Multifocal PVCs** will occur if more than one ectopic area begins to initiate early ventricular beats. For example, if three ectopic ventricular sites began initiating PVCs, each site would produce a different looking PVC waveform. The ECG criteria are basically the same as unifocal PVCs. Multifocal PVCs are considered more dangerous when compared to unifocal PVCs, as this represents a greater amount of myocardial irritability.

*Sample of a patient in NSR with a couplet of PVCs from two foci (multifocal).*

![Sample ECG showing multifocal PVCs](image)

PVCs may also occur in succession. When this happens, the PVCs are called a **couplet**. (The strip above also shows a couplet.) The term **ventricular bigeminy** is used for a grouped beating pattern when every other beat is a PVC (despite the underlying rhythm). For example, ventricular bigeminy is a when you see a pattern of one PVC, then one normal beat, then one PVC, followed by a normal beat.

**Section 13: Premature Ventricular Contractions**
If every other beat is a PVC, ventricular bigeminy is present. If every third beat is a PVC, the term **ventricular trigeminy** is used; if every fourth beat is a PVC, **ventricular quadrigeminy** is present; and so forth.

*This strip is an example of ventricular trigeminy.*

![Sample ECG showing ventricular trigeminy](image)

Keep in mind, PVCs may occur as isolated complexes, or they may occur repetitively in pairs (two PVCs in a row). When three or more PVCs occur in a row, whether unifocal or multifocal, **ventricular tachycardia** (VT) is present. When VT lasts for more than 30 seconds, it is arbitrarily defined as **sustained ventricular tachycardia**.

**R on T Phenomenon:**
The T wave is a sensitive or vulnerable area in the cardiac electrical cycle. Remember that the heart is now repolarizing and does not like to be stimulated at this time. If an early ventricular beat comes in on top of or near the T wave, the early beat could throw the heart into an uncontrollable repetitive pattern called ventricular tachycardia. The term **“R on T phenomenon”** is used whenever an early ventricular beat lies near the vulnerable T wave. Consult with MD if you see early R waves coming in near the T wave. Early detection can help prevent your patient from developing a life-threatening rhythm (Heitman et al., 2012; Jacobson, et al., 2014).
Possible Causes of PVCs:
- Caffeine, tobacco, alcohol
- Digoxin toxicity
- Exercise
- Hypocalcemia
- Hyperkalemia
- New MI
- Proarrhythmic effect of antiarrhythmic agents

ECG Criteria:
1. **Heart rate**: Depends on the underlying rhythm.
2. **Rhythm**: Depends on the underlying rhythm. The PVC beats are premature, so this will make the R to R interval a bit irregular.
3. **P waves**: Not applicable (there are no P waves associated with PVCs).
4. **PR interval**: Not applicable.
5. **QRS width**: > 0.12 seconds, wide and bizarre in appearance. T wave may be opposite direction of QRS complex.

Nursing Priorities:
- Assess the patient’s response.
- Many patients are asymptomatic, while others may feel palpitations or light-headed.

Potential Treatments:
Treatment is required only when PVCs are frequent or the patient has intolerable symptoms.
- **Asymptomatic**: Observation. Rule out hypokalemia and hypoxemia (both can trigger PVCs). Oxygen. Correct electrolyte imbalances.
- **Symptomatic**: In the setting of an acute myocardial infarction, PVCs indicate the need to aggressively treat the ischemia/infarction with oxygen, nitroglycerin, morphine, and potential antiarrhythmic agents (Heitman et al., 2012; Jacobson, et al., 2014).

Section 13: Ventricular Tachycardia

Description
Ventricular tachycardia (VT) is defined as three or more consecutive PVCs in a row at a rate greater than 100 beats per minute. VT is generally caused by single foci in either ventricle that fire at a rapid rate to override the SA node and thereby take control of the heart’s rhythm. A short run of consecutive PVCs is often called a “burst” of VT.

ECG characteristics include a rapid, regular rhythm with a wide QRS. The QRS is wide since the origin of the rhythm is outside the bundle branches, thereby taking a longer time to conduct cell to cell
within the ventricle. Ventricular tachycardia may be **monomorphic** (all QRSs with the same shape) or **polymorphic** (varying QRS shapes during the tachycardia).

Atroventricular dissociation is present, but not always noticeable. This means that the sinus node is depolarizing the atria in a normal manner at a rate either equal to or slower than the ventricular rate. Thus sinus P waves sometimes can be recognized between QRS complexes but do not conduct down into the ventricles.

This arrhythmia may be either well tolerated or associated with life-threatening hemodynamic compromise. The hemodynamic consequences of VT depend largely on the presence or absence of myocardial dysfunction (such as might result from ischemia or infarction) and on the rate of VT (the faster the rate, the less well tolerated).

**Possible Causes:**
- CAD
- Drug toxicity
- Electrolyte imbalance
- Myocardial irritability
- Acute MI
- Heart failure (Heitman et al., 2012; Jacobson, et al., 2014).

**ECG Criteria:**
1. **Heart rate**: 100-250 bpm.
2. **Rhythm**: Ventricular rhythm regular.
3. **P waves**: P waves may or may not be seen. If present, they are not associated with the QRS complex. (AV disassociation occurs with this rhythm, but P waves are not always seen.)
4. **PR interval**: Not applicable.
5. **QRS width**: > 0.12 seconds, wide and bizarre in appearance. Difficult to differentiate between the QRS and the T wave.

**Nursing Priorities:**
- Check your patient’s pulse and blood pressure to determine if this is stable, unstable (systolic blood pressure <90mmHg) or pulseless VT.
- Notify physician.
- Treatment depends on the patient’s response to the VT rhythm (Heitman et al., 2012; Jacobson, et al., 2014).

**Potential Treatments:**
- **Asymptomatic or stable**: Oxygen, obtain 12-lead ECG, anti-arrhythmics (amiodarone as first choice). Check electrolytes such as potassium and magnesium. Cardioversion may be necessary if patient becomes unstable.
- **Symptomatic**: If unstable, prepare for immediate synchronized cardioversion. Consider amiodarone bolus and infusion. If pulseless, administer one defibrillation shock followed by CPR. Activate your code team and follow ACLS guidelines (Heitman et al., 2012; Jacobson, et al., 2014).
Section 13: Ventricular Fibrillation

Description

Ventricular fibrillation (VF) is the result of highly irritable ventricle(s), which begin to send out rapid electrical stimuli. The stimuli are chaotic resulting in no organized ventricular depolarization. The ventricles do not contract because they never depolarize. For you visual learners... just imagine shaking a bowl full of Jell-O. Gently shake the bowl and watch the Jell-O quiver or “fibrillate.” This is similar to what is happening within the heart. Because the ventricles are fibrillating and never contracting, the patient does not have a pulse, cardiac output, or blood pressure.

The terms coarse and fine have been used to describe the amplitude of the waveforms in VF. With coarse VF, the fibrillatory waves are more easily seen and are usually greater than 3mm in height (3 small boxes tall). Coarse VF usually indicates a more recent onset of VF, which could be more easily converted by prompt defibrillation. The presence of fine VF (which looks a bit like asystole and is less than 3mm in height) often means there has been a considerable delay since collapse, and successful resuscitation is more difficult.

Possible Causes:

- Acute MI.
- Untreated ventricular tachycardia.
- Underlying heart disease.
- Acid-base imbalance.
- Electrolyte imbalances such as hypokalemia, hyperkalemia, and hypercalcemia (Heitman et al., 2012; Jacobson, et al., 2014).
Section 13: Ventricular Fibrillation

ECG Criteria:
1. **Heart rate**: None. No discernable P waves or QRS complexes.
2. **Rhythm**: Chaotic wavy recording. No discernable rhythm.
3. **P waves**: None.
4. **PR interval**: Not applicable.
5. **QRS width**: Not applicable.

Nursing Priorities:
Check for an airway, breathing, and pulse per Basic Life Support (BLS) standards. Call for help. The patient will be apneic, pulseless, and unresponsive. Begin CPR. VF treatment requires defibrillation. Resuscitation requires defibrillation and emergency drugs per ACLS VF guidelines. The sooner the patient is defibrillated, the greater likelihood of achieving spontaneous circulation. The longer the patient is in VF, the more difficult it is to convert the rhythm.

Potential Treatments:
Initial treatment is always defibrillation. Only defibrillation provides definitive therapy. Other priorities include securing an airway, making sure the patient has IV access, and administering medications per guidelines (Heitman et al., 2012; Jacobson, et al., 2014).

Section 14: Asystole

Description
Asystole represents the total absence of electrical activity. Since depolarization does not occur, there is no ventricular contraction and a straight line will appear on the ECG. To assure there is no electrical activity, check your patient's pulse first, but also check the rhythm in a second lead and make sure your monitor is working properly. Asystole may occur as a primary event in cardiac arrest, or it may follow VF.

In addition, the distinction between very fine VF and asystole may be very difficult. If it might be VF, it should be treated with defibrillation. If no organized QRS complex is seen and the patient has a pulse, then the ECG is improperly connected, turned off, or improperly calibrated.

Possible Causes:
- Severe metabolic deficit.
• Acute respiratory failure.
• Extensive myocardial damage or ruptured ventricular aneurysm (Heitman et al., 2012; Jacobson, et al., 2014).

Nursing Tip:
Assess your patient! Treat the patient and not the monitor!

ECG Criteria:
1. Heart rate: None
2. Rhythm: None
3. P waves: None
4. PR interval: None
5. QRS width: None

Nursing Priorities:
Check rhythm in a second lead (make sure a lead has not fallen off). If your patient has a pulse, they are obviously NOT in asystole. If the patient is pulseless, initiate CPR and call for help.

Potential Treatments:
Continue CPR and secure airway and IV access. Search for possible causes. Implement medication therapy per ACLS guidelines (Heitman et al., 2012; Jacobson, et al., 2014).

Test Yourself
Interpretation of the rhythm displayed is:

1. Ventricular fibrillation
2. Sinus rhythm with multifocal PVCs - Correct
3. Ventricular tachycardia
Section 15: Pulseless Electrical Activity

Description
It is possible for a patient to have what appears to be a normal or somewhat normal rhythm, but no pulse (and therefore no circulation). Pulseless electrical activity (PEA) is a cardiac rhythm in which the heart has some type of electrical activity but there is no palpable pulse. Without a pulse there is no perfusion.

PEA can appear on the monitor as any somewhat organized electrical activity; however your patient will be unconscious, pulseless, and apneic. PEA provides an excellent example of the rationale for checking for a pulse rather than interpreting a rhythm from looking at a monitor.

Nursing Tip:
Call your code team for any patient that is apneic and or pulseless, regardless of the rhythm presented on the monitor.

Possible Causes:
- Hypovolemia
- Cardiac tamponade
- Hypoglycemia
- Hypoxia
- Hypothermia
- Hyper/hypokalemia
- Trauma
- Toxins (i.e. drugs)
- Tension pneumothorax
- Thrombosis (cardiac or pulmonary) (Heitman et al., 2012; Jacobson, et al., 2014)

ECG Criteria
1. Any somewhat organized electrical activity without a pulse.

Nursing Priorities:
Follow the ABCs! Perform effective CPR and ensure that you have an open airway. Provide supplemental oxygen. IV access is critical in order to provide medications and fluids as necessary.

Potential Treatment:
Once an IV is established, plan to administer a vasopressor such as epinephrine or vasopressin. Consider atropine for a slow PEA rate. Continue CPR and treat PEA according to contributing factors.

Since the causes of PEA are variable, treatment is variable. If the patient has cardiac tamponade, relief of the pressure on the heart will be a priority. If the patient is hypovolemic, fluid administration will be a key priority (Heitman et al., 2012; Jacobson, et al., 2014).
Section 16: Types of ECG Recording Interference

Because the complexes seen on the ECG recording are a reflection of electrical activity, other interfering factors can distort the quality of the tracing. The term artifact is used when an ECG recording is distorted by non-cardiac factors. **Common causes of interference or artifact include:**

- Muscle movement, shivering or seizure activity.
- Loose electrodes.
- Patient movement.
- 60 cycle interference (the influence of other equipment in the room which is improperly grounded).

**Practice Rhythm Strip #1**

The following rhythm strips are for your practice. We suggest you practice with these prior to taking the post test.

*(All strips are six-second strips unless otherwise indicated.)*

**Rhythm Strip #1 Answer**

- **Heart rate:** 60
- **Rhythm:** Regular
- **P waves:** One present for each QRS
- **PR interval:** .20
- **QRS width:** .08

**Interpretation:** Normal sinus rhythm 1st degree AV block
**Practice Rhythm Strip #2**

**Rhythm Strip #2 Answer**

Heart rate: 100  
Rhythm: Regular  
P waves: One present for each QRS  
PR interval: .14  
QRS width: .08  
Interpretation: Sinus rhythm/sinus tachycardia

**Practice Rhythm Strip #3**

**Rhythm Strip #3 Answer**

Heart rate: 40  
Rhythm: Regular  
P waves: One present for each QRS  
PR interval: .12  
QRS width: .08  
Interpretation: Sinus bradycardia with (unifocal) ventricular bigeminy
Practice Rhythm Strip #4

Heart rate: 78
Rhythm: Regular
P waves: Absent
PR interval: Not applicable
QRS width: .08
Interpretation: Atrial flutter

Practice Rhythm Strip #5

Heart Rate: 40
Rhythm: Regular
P waves: More than one present for each QRS
PR Interval: For the conducted beats it is fixed at .14
QRS Width: .08
Interpretation: Second degree heart block Type II
Rhythm Strip #6 Answer

**Heart rate:** 150  
**Rhythm:** Irregular  
**P waves:** Absent  
**PR interval:** Not applicable  
**QRS width:** .08  
**Interpretation:** Uncontrollable atrial fibrillation

Rhythm Strip #7 Answer

**Heart rate:** 70  
**Rhythm:** Atrial rate is regular, ventricular rate is irregular  
**P waves:** More than one present for each QRS  
**PR interval:** Progressively longer until one P is not followed by a QRS. After the blocked beat the cycle starts again.  
**QRS width:** .08  
**Interpretation:** Second degree heart block Type I
**Practice Rhythm Strip #8**

<table>
<thead>
<tr>
<th>ECG Criteria:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Heart rate:</strong> 40</td>
</tr>
<tr>
<td><strong>Rhythm:</strong> P to p is regular but not associated with one another</td>
</tr>
<tr>
<td><strong>P waves:</strong> More than one present for each QRS</td>
</tr>
<tr>
<td><strong>PR interval:</strong> No PR interval</td>
</tr>
<tr>
<td><strong>QRS width:</strong> .10</td>
</tr>
<tr>
<td><strong>Interpretation:</strong> Third degree (complete) heart block</td>
</tr>
</tbody>
</table>

**Rhythm Strip #9**

<table>
<thead>
<tr>
<th>ECG Criteria:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Heart rate:</strong> 130</td>
</tr>
<tr>
<td><strong>Rhythm:</strong> Regular</td>
</tr>
<tr>
<td><strong>P waves:</strong> Hidden</td>
</tr>
<tr>
<td><strong>PR interval:</strong> Not applicable</td>
</tr>
<tr>
<td><strong>QRS width:</strong> Wide and bizarre</td>
</tr>
<tr>
<td><strong>Interpretation:</strong> Ventricular tachycardia</td>
</tr>
</tbody>
</table>
Practice Rhythm Strip #10

Rhythm Strip #10 Answer

Heart rate: None (Initial sinus rhythm)
Rhythm: Irregular
P waves: Initially one present per QRS then absent
PR interval: Initially .14, then not applicable
QRS width: Initially .10, then absent/not applicable Interpretation: Sinus rhythm into ventricular tachycardia, into ventricular fibrillation

Practice Rhythm Strip #11

Rhythm Strip #11 Answer

Heart rate: None
Rhythm: Absent
P waves: Absent
PR interval: Not applicable
QRS width: None
Interpretation: Asystole
Practice Rhythm Strip #12

Rhythm Strip #12 Answer

Heart rate: 70
Rhythm: Regular
P waves: One present for every QRS
PR interval: .28
QRS width: .08
Interpretation: Normal sinus rhythm, 1st degree AV block

Practice Rhythm Strip #13

Rhythm Strip #13 Answer

Heart rate: 72
Rhythm: Regular with ectopic beats
P waves: One present before each QRS except when ectopic beats occur
PR interval: .20
QRS width: .08
Interpretation: Normal sinus rhythm with PVC's; a ventricular couplet and then 4 beat run of V-Tach
Practice Rhythm Strip #14

Rhythm Strip #14 Answer

Heart rate: 26
Rhythm: Regular
P waves: One present before each QRS
PR interval: .16
QRS width: .06
Interpretation: Sinus bradycardia

Conclusion
This module described the anatomy, physiology, and basic electrophysiologic knowledge needed to understand basic ECG interpretation. A six-step process of rhythm identification is used as a framework for the interpretation of rhythms originating in the sinus, junctional, ventricular areas. Heart blocks and life threatening rhythms were also discussed. Beyond interpretation, all rhythms include nursing priorities and potential treatment strategies.

Every attempt has been made to provide information that is consistent with current literature including the American Heart Association guidelines. Mastery of this complex topic requires several months to years of practice in the clinical setting. Many first time learners need to re-review sections of this module as their learning develops and when new questions arise.

Additional Resources
For further information, the following web sites offer you the opportunity to practice ECGs with strips from their websites. Keep in mind that with ECG interpretation “practice, practice, practice” is key to making this information “stick.”

University of Utah-School of Medicine.
Alan E. Lindsay ECG Learning Center
http://ecg.utah.edu/

ECG Library
http://www.ecglibrary.com/ecghome.php

American Heart Association
http://www.heart.org/HEARTORG/
Appendix: Bundle Branch Blocks

In the healthy heart, the right bundle branch quickly conducts the stimulus of depolarization to the right ventricle and the left bundle branch conducts to the left ventricle. The depolarization stimulus is nearly simultaneous and conduction to both ventricles occurs at nearly the same time. This simultaneous conduction is recorded in a QRS complex that is ≤.12 seconds.

A block in one of the bundle branches produces a delay of depolarization to its ventricle. The unblocked bundle branch will depolarize before the blocked side. Since the blocked bundle branch can no longer conduct an impulse, this ventricle must depolarize in a slower, cell-to-cell method (the ventricle does eventually depolarize, but the cell-to-cell method is less efficient and slower). Due to this slowed process, the QRS width is greater than .12 seconds in the setting of a bundle branch block. Some physicians and nurses call the widened QRS an “intraventricular conduction delay (IVCD)” which is a term used interchangeably with the term “bundle branch block.”

Characteristics of the QRS may vary a bit. Have you ever heard the saying “rabbit ears”? As you know, the widened QRS represents the non-simultaneous depolarization of both ventricles. The ECG might record this delay with one wide R wave or with two separate R waves. Two separate R waves might be called “rabbit ears” as they resemble two pointed ears on a rabbit head. The two separate R waves are really just a recording of the left and right ventricles depolarizing at different times (Jacobson, Marzlin, & Webner, 2014).

If the right bundle is blocked, it is termed a Right Bundle Branch Block. If the left bundle is blocked, it is called a Left Bundle Branch Block. Bundle branch blocks (BBB) are reflective of damage to one of the bundles. It is most often associated with coronary artery disease. Once a person develops a widened QRS, they are likely to keep this for life. Most BBB are stable and often do not require treatment other than observation and measurement of the QRS width. Notify the MD if the width is increasing. In the setting of an acute MI, a new bundle branch block (or widening QRS) may reflect newly damaged myocardium and the potential need for a temporary pacemaker.
Caution… Do not let the wide QRS fool you for ventricular ectopy. The person with a BBB will have a P wave before the widened beat AND the QRS width will always be wide. The person with ventricular ectopy will not have a P wave preceding the widened beat and only the ectopic beats will be >.12 seconds.

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