ACKNOWLEDGEMENTS

RN.com acknowledges the valuable contributions of...

...Tracey L. Noble, RN, BSN, the author of *Lethal Arrhythmias*. Tracey earned her BSN at the University of Pennsylvania in Philadelphia, Pennsylvania. She started her nursing career at New York University Medical Center in Manhattan, New York on an adult medical unit. After spending most of her time in the cardiac step-down unit, Tracey realized her love of the cardiac patient.

Since then, Tracey has worked at the bedside in an intermediate care unit that specializes in the adult cardiac patient in San Diego, California and in the Heart Center at Salinas Valley Memorial Hospital in Salinas, California. As a clinical instructor for Hartnell College, Tracey instructed nursing students in adult cardiac medical/surgical care.

Tracey currently works in cardiac care as an Advanced Clinician in an intermediate care unit at Sharp Memorial Hospital in San Diego, California. As an Advanced Clinician, Tracey's love for patient care meets with her passion for teaching.

...Tanna R. Thomason, RN, MS, CCRN, contributor to this course. Tanna is the primary author of RN.com’s *ECG Interpretation: Learning the Basics*. Tanna has over 20 years of experience as a clinician in the hospital setting. After completing her Master’s Degree as a Clinical Nurse Specialist from San Diego State University in 1993, Tanna functioned as a critical care Clinical Nurse Specialist for Sharp Memorial Hospital in San Diego, CA. In addition to her Clinical Nurse Specialist role, Tanna has been teaching nursing students since 1998 in an adjunct faculty position at Point Loma Nazarene University. In 2001, Tanna became President of Smart Med Ed, an educational consulting business. Tanna is a member of the American Association of Critical Care Nurses (AACN) and has served in various leadership roles for the San Diego Chapter of AACN. Other memberships include Sigma Theta Tau and the Cardiovascular Council of the American Heart Association.

...Karen Siroky, RN, MSN, for the 2005 review and update of this course. Karen is the Director of Education for RN.com. Karen received her B.S.N. from the University of Arizona and her MSN from San Diego State University. Her nursing experience includes ICU, transplant coordination, recruitment, quality improvement, information, and education. She has previously published articles on Quality Improvement and Cardiac Transplantation.
**PURPOSE & OBJECTIVES**

The purpose of *Lethal Arrhythmias: Advanced Rhythm Interpretation* is to instruct nurses on the identification and initial treatment of lethal arrhythmias. This course will build a foundation for recognizing and preventing these rhythms by providing a basic understanding of ECG rhythm interpretation. Case studies will help you apply what has been learned.

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

1. Discuss the basic electrophysiology of the heart.
2. Interpret an ECG waveform.
3. Discuss the importance of recognizing and identifying premature ventricular contractions.
4. State the possible causes for premature ventricular contractions.
5. State the warning signs of lethal arrhythmias.
6. Recognize and identify seven lethal arrhythmias.
7. State the possible causes for the lethal arrhythmias.
8. Describe the initial treatment for the seven lethal arrhythmias.
9. Articulate how to use a cardiac defibrillator/external pacemaker for cardioversion, defibrillation, and transcutaneous pacing.
INTRODUCTION

As nurses, we need to be prepared in the event of a lethal arrhythmia: a deadly heart rhythm. When a patient suffers a lethal arrhythmia every few seconds count. Are you ready? Will you be able to make the quick decisions necessary to save your patient's life? Although patients with known cardiac disease suffer the greatest number of lethal arrhythmias, these rhythms can occur with any patient on any floor.

In this course, you will study ventricular arrhythmias and lethal rhythms. You will learn about Premature Ventricular Contractions, Ventricular Tachycardia, Ventricular Fibrillation, Pulseless Electrical Activity, Agonal Rhythms, and Asystole. You will learn how to detect the warning signs of these rhythms, how to quickly interpret the rhythm, and to prioritize your nursing interventions. The different treatments for each of these rhythms will be discussed as well as the different defibrillators that are currently being used.

The course begins with a brief overview of the heart, the conduction system and rhythm interpretation. If you are not proficient in these areas, we recommend you take our introductory course, ECG Interpretation: Learning the Basics, before taking this course.
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 blood passes through the tricuspid valve to the right ventricle. The right ventricle pushes the oxygen poor blood through the pulmonic valve into the pulmonary arteries and then to the lungs. The blood is then oxygenated in the lungs.

The left atrium receives oxygen-rich blood returning from the lungs through pulmonary veins. The mitral valve separates the left atrium and left ventricle. The left ventricle pushes the oxygen-rich blood through the aortic valve and into 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.

Heart Sounds and Atrial Kick

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.

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).
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 **electrical (conductive) cells** of the heart initiate electrical activity.
- 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. Specific mechanical **(contracting) cells** respond to the stimulus and of the electrical cells and contract to pump blood.

**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.

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 conduct again.

The following picture represents normal cardiac rhythm and how it relates to the depolarization and repolarization of the cardiac cells.
THE CARDIAC 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. 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. Let us now discuss each structure in more detail.

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. 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.

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.

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. The AV node and the bundle of His are referred to collectively as the AV junction. The Bundle of His conducts the electrical impulse down to the right and left bundle branches. The bundle branches further divide into Purkinje fibers.
**Purkinje Fibers**

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**. 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.

A more comprehensive review of the conduction system is included in our course *ECG Interpretation: Learning the Basics*. If you still have questions about how the conduction system of the heart works, please refer to that course or other references.
ECG WAVEFORMS

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.

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. 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. 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.

The diagram above illustrates the configuration of ECG graph paper and where to measure the components of the ECG waveform.

Normal ECG Waveforms and Intervals

As this course is geared toward advanced practitioners, standard lead selection, placement and tracings are not contained in this course. If you need to review these, please refer to other textbooks or our course ECG Interpretation: Learning the Basics.

To evaluate an ECG rhythm strip, it is standard practice to print a strip of at least a 6-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.

When an ECG strip is printed, most printers provide heart rate information at the top of the strip. Nevertheless, 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. We will briefly review three of the most common methods for heart rate calculation.
**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 6-second strip Count the number of R waves in a 6-second strip and multiply by 10. For example, if there are seven (7) R waves in a 6-second strip, the heart rate is approximately 70 or 7x10=70.

**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.

**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.

Remember, if you only have a short rhythm strip (< 6 seconds), you will need to use either the Large Box Method or the Small Box Method.
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 6 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.

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 and ST/ T wave shape and size
6. Rhythm 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.

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

Step 2 is the assessment of the regularity of the rhythm. Is the rhythm regular or is it irregular?

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**.

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.

<table>
<thead>
<tr>
<th>Normal Findings:</th>
<th>Abnormal Findings:</th>
</tr>
</thead>
<tbody>
<tr>
<td>♦ The R-R intervals are regular.</td>
<td>♦ The R-R intervals are irregular.</td>
</tr>
<tr>
<td>♦ The P-P intervals are regular.</td>
<td>♦ The P-P intervals are irregular.</td>
</tr>
<tr>
<td>♦ There is one P for every QRS.</td>
<td>♦ There is more than one P for each QRS.</td>
</tr>
</tbody>
</table>

**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.

<table>
<thead>
<tr>
<th>Normal Findings</th>
<th>Abnormal Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>♦ P waves should be regular (march out the P-P intervals with your calipers).</td>
<td>♦ A P wave is not followed by a QRS complex.</td>
</tr>
<tr>
<td>♦ P waves have a symmetrical shape, usually upright and rounded.</td>
<td>♦ There are more P waves than QRS complexes (There should be one P for every QRS or a 1:1 relationship.)</td>
</tr>
<tr>
<td>♦ P waves should all look alike (uniform) and should point in the same direction.</td>
<td></td>
</tr>
</tbody>
</table>

*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.
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.

### Normal Findings:
- The PR interval (or time travel from SA to AV nodes) is between **0.10 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).

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 the S wave. Normal ventricular conduction and depolarization takes no more than 0.10 seconds.

### 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 >0.10 seconds wide.
- The R to R interval between each QRS is irregular.
**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 of the R wave.

**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. Current treatments for each type of ECG rhythm are presented, but keep in mind that the science of cardiology is changing quickly and your hospital may have different standards.
PREMATURE VENTRICULAR CONTRACTIONS

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.

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.” 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 (0.10 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.

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 a unifocal PVC.
**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 slightly 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. Below is a sample of a patient in NSR with a couplet of PVCs from two foci (multifocal).

![ECG Sample](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.

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. The strip below is an example of ventricular trigeminy.

![ECG Sample](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.

**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:** > 0.10 seconds, wide and bizarre in appearance. T wave may be opposite direction of QRS complex
6. **Rhythm Interpretation:** Normal Sinus Rhythm with PVCs
**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.

**Common Causes of PVCs**

Let's examine some of the common causes of PVCs – The causes of PVCs are critical as PVCs often lead to other lethal arrhythmias.

- **Acute Myocardial Ischemia**  
- **Electrolyte Imbalances**  
- **Hypoxemia**
- **Acid/Base Imbalances**  
- **Mechanical Disturbances**  
- **Cardiomyopathy**
- **Digitalis Toxicity**  
- **Other Pharmacologic Agents**  
- **Hypothermia**

**Acute Myocardial Ischemia**

Acute Myocardial Ischemia is the most common cause of PVCs that leads to lethal arrhythmias. "When ischemic tissue loses its ability to control the movement of potassium and sodium across the cell membrane, ventricular arrhythmias, like Ventricular Tachycardia (VT) and ventricular fibrillation (VF), may occur. Cardiac ischemia also may damage the heart's electrical conduction fibers, delaying or completely blocking the electrical impulses of the sinoatrial node" (Davenport & Morton, 1997). This is the most dangerous of all the causes of PVCs.

**Electrolyte Imbalances**

Potassium, Calcium, and Magnesium are the three electrolytes most commonly associated with ventricular ectopy. If these electrolytes are not in perfect balance, they predispose the cell membranes of the myocardial tissue to instability. All of these imbalances may cause other ECG changes and symptoms in addition to PVCs.

Normal Potassium (K+) serum level is 3.8-5.0 mEq/liter

**Hypokalemia**

Hypokalemia has other tell tale signs that can be discovered on the ECG. Look for ST segment depression, flattened or inverted T waves or a small upright wave that follows the T wave, called a U wave. Your patient may experience shallow respiration, a slow weak pulse, abdominal cramps, confusion, drowsiness, muscle weakness or tenderness, paralytic ileus, postural hypotension, increased urine output, and nausea and vomiting. Hypokalemia is treated with potassium supplements, either by mouth or IV.

**Hyperkalemia**

Hyperkalemia, an elevated potassium level, can be detected on the ECG by tall peaked T waves. Your patient may experience irritability, anxiety, abdominal cramping, diarrhea, mental confusion, intestinal colic, weakness (especially of the lower extremities), and parasthesia (abnormal sensations such as burning or pricking) (Tortora & Grabowski, 1993). Hyperkalemia is treated with "IV calcium gluconate to stabilize the myocardium, IV sodium bicarbonate to correct the acidemia, or a glucose and insulin infusion to drive extracellular potassium back into cells" (Davenport & Morton, 1997). Non-potassium sparing diuretics and Kayexalate are also used. If these treatments fail or if the patient is in renal failure, dialysis may be necessary.
Ca++ Normal serum calcium (Ca++) levels are 8.5 - 10.5 mg/dL

Hypocalcemia
With Hypocalcemia, the EKG shows a lengthening of the ST segment and the QT interval. Your patient may experience numbness and tingling of the fingers, tingling around the mouth, hyperactive reflexes, abdominal cramps, muscle cramps, tetany, laryngeal muscle spasms, bone fractures, vomiting, diarrhea, and seizures. Hypocalcemia is treated with oral Calcium supplements and IV Calcium. Vitamin D supplements are also used in conjunction with the oral Calcium.

Hypercalcemia
Hypercalcemia is noted on the EKG by shortened QT intervals and lengthened QRS complex. Your patient may experience fatigue, muscle hypotonicity, drowsiness, disorientation, anorexia, nausea, vomiting, polyuria, itching, confusion, paresthesia, stupor, constipation, kidney stones, thirst, bone pain, and even coma due to hypercalcemia. Saline infusions along with Lasix are often used to treat hypercalcemia. IV Phosphate may also be used to cause a decrease in the serum Calcium (Tortora & Grabowski, 1993 and Davenport & Morton, 1997).

Mg++ Normal Magnesium (Mg++) levels are 1.5-2.1 mEq/L

Hypomagnesemia
Hypomagnesemia is often seen on the ECG by a prolonged QT interval and a broadened T wave. Your patient may experience muscle weakness, tremors, vertigo, ataxia, irritability, delirium, convulsions, anorexia, nausea, and confusion (Tortora & Grabowski, 1993 and Davenport & Morton, 1997). Hypomagnesemia is treated with IV Magnesium.

Hypermagnesemia
"Elevated levels of serum Magnesium aren't usually hazardous until the serum is four times the normal value of 1.5 - 2 mEq/L - and even at those levels, it doesn't produce VT or VF" (Davenport & Morton, 1997).

Causes of Electrolyte Imbalances
The causes of electrolyte imbalances are many and varied. It is critical for you to be familiar with these causes in order to prevent PVCs. Some of the most common causes follow: various types of diuretics, vomiting, diarrhea, renal failure, dehydration, malnutrition, Crohn’s disease, and sepsis.

O2 Normal PaO2 value is 75-100 mm Hg

Hypoxemia
Hypoxia is insufficient oxygenation of the blood and can cause irritability of the myocardium. "The ability of oxygen values to change very rapidly in a critically ill patient must not be underestimated" (Thelan, et al., 1998). Treatment is aimed at increased oxygenation. Besides PVCs, the ECG may show inverted T waves or ST elevation when the patient is hypoxemic.

Causes of Hypoxemia
- Chronic anemia
- Coronary artery disease
- Narcotics
- Chest tube placement
- Sedation
- Pulmonary embolism
- Chronic obstructive pulmonary disease
Metabolic Acidosis

Metabolic acidosis is excess serum Hydrogen in the presence of low pH. Your patient may experience lethargy, drowsiness, headache, nausea, vomiting, diarrhea, and abdominal pain. Treatment will be aimed at increasing the pH. IV Sodium Bicarbonate is the drug of choice. IV fluids and Oxygen will also be ordered.

Causes of Metabolic Acidosis

- Renal disease
- Severe dehydration
- Diarrhea
- Vomiting
- Impaired liver function
- Intestinal malabsorption
- Starvation
- Ingestion of acids or acid salts
- Ketosis

(Thomas, 1993; Davenport & Morton, 1997, p. 53)

Hypothermia

A core body temperature below normal is considered hypothermia. "Hypothermia can destabilize myocardial conduction by increasing oxygen consumption - initially, through shivering, and in later stages, through vasoconstriction" (Davenport & Morton, 1997). Hypothermia also inhibits the Na⁺ pump, causing an accumulation of intracellular Na⁺ (Marriott & Conover, 1998). A core body temperature below 30° C or 86° F causes all bodily processes to slow down. Besides PVCs, the ECG will then show a prolongation of the PR, QRS and the QT.

Cardiomyopathy

Cardiomyopathy is disease of the myocardium. The disease refers to the structural and functional aspects of the heart. Cardiomyopathy is broken down into three different categories:

- Dilated Cardiomyopathy
- Hypertrophic Cardiomyopathy
- Restrictive Cardiomyopathy

In dilated cardiomyopathy, the heart enlarges and the ventricles become hypertrophied. This leads to a decrease in cardiac output, which predisposes the myocardium to arrhythmias. In hypertrophic cardiomyopathy, the left ventricle becomes hypertrophied and hypercontractile. In restrictive cardiomyopathy, the myocardium becomes thick and fibrotic. Ventricular arrhythmias are more common in the first two types of cardiomyopathy.

Your patient with cardiomyopathy may experience shortness of breath, chest pain, edema, intolerance of activity, dizziness, and fatigue. Treatment may include diuretics, beta-blockers, Ca++ channel blockers, ACE inhibitors, anticoagulants, and antiarrhythmics. Oxygen therapy and fluid management will also be included. In some cases, cardiac transplant may be necessary. Internal cardiac defibrillators are common in this patient population.

In addition to PVCs, the ECG may reveal a widened QRS complex or prolonged PR interval.
**Digitalis Toxicity**

Digoxin is a cardiac glycoside used to treat congestive heart failure and various atrial arrhythmias. Digoxin increases intracellular calcium, thus increasing the force of contraction, decreases the heart rate and decreases AV node conduction (Skidmore-Roth, 2005). “Some drugs - like quinidine, amiodarone, and calcium channel blockers - increase the effects of Digoxin and, as a result, increase the risk of Digoxin toxicity. Hypokalemia also potentiates Digoxin toxicity” (Davenport & Morton, 1997).

Digoxin toxicity is a serum Digoxin level >2.1 ng/ml.

With digoxin toxicity, the ECG will reveal a shortened QT interval. Your patient may experience malaise, visual distortion, vomiting, dizziness, hyperventilation and delirium (Davenport & Morton, 1997). Treatment includes stopping the Digoxin, correcting electrolyte imbalances, evaluating the use of other prescribed drugs that may have precipitated the effect of the Digoxin and providing temporary pacing of the heart (Davenport & Morton, 1997). If the serum level is greater than 10 ng/ml, Digibind, an antidote for digitalis toxicity may be ordered.

**Vision Changes in Your Patient?** This may be a clue to Digoxin toxicity. Yellow vision or hazy vision may be noted. Listen to your patient and/or ask about vision changes. Include this information in patient teaching.

**Other Pharmacologic Agents**

Antidysrhythmic drugs that are prescribed to treat arrhythmias may actually cause lethal arrhythmias. Quinidine, procainamide (Pronestyl), disopyramide (Norpace), sotalol (Betapace), amiodarone (Cordarone), and propafenone (Rythmol), are among these drugs. These drugs cause an increase in the QT interval. Patients started on these medications should be placed on cardiac monitoring, and you should measure the QT interval every shift. If the QT interval increases by 25% from the baseline established prior to administering the drug, the physician should be notified and drug should be stopped.

There are other drugs that are prescribed for your patients that also may increase the QT interval and put your patients at risk for lethal arrhythmias (Thaler, 2003). These medications include tricyclic antidepressants, antifungal drugs, some antihistamines, and erythromycin.
**Mechanical Disturbances**

A physical irritation of the myocardium may cause PVCs. The insertion of a central venous catheter, Swan-Ganz catheter, or a cardiac catheterization catheter is the means of mechanically irritating the heart. Cardiac surgery may also mechanically cause PVCs.

All of these factors can cause PVCs that may lead to other lethal arrhythmias. Before we move on, let us review what you will do if you suspect PVCs:

- ♦ Assess your patient.
- ♦ Look for changes in the mental status.
- ♦ Look for changes in the blood pressure.
- ♦ Check the apical pulse or other signs.
- ♦ Check O2 saturation.
- ♦ Note any changes in skin color, temperature, or decreased urine output.

Thoroughly document your findings. Run a rhythm strip. If you are not on a floor where heart monitors are available, any of the cardiac defibrillators will allow you to run a rhythm strip. If PVCs are found, report them to the physician. The physician will likely draw serum electrolytes, specifically K⁺, Ca+++, and Mg++. Also, ask the physician to draw an ABG to check the pH and the PaO₂ level. Your unit may have standing orders to follow. If any of the above factors can be corrected, now is the time to do so! Your assessment skills and early intervention may save a life!

---

*NURSING TIP:* If your patient is not on a cardiac monitor, how do you assess for possible PVCs? Listen to the apical heart rate for one minute. It may reveal an irregular heart rhythm. Watch for a decrease in your patient's level of consciousness, a decrease in your patient's urine output, a decrease in your patient's skin temperature or a loss of color, and a change in your patient's blood pressure. SUSPECT PVCs! Question whether your patient needs to be on a cardiac monitor or perhaps transferred to a telemetry unit!
Your patient may or may not be symptomatic with PVCs. Your patient may or may not complain of palpitations. Some patients do not even know they are experiencing PVCs. However, PVCs do cause decreased filling time and decreased stroke volume, which lead to decreased cardiac output. This leads to decreased perfusion to the vital organs.

**Nursing Interventions**

Here are your red, alert warning signs for PVCs that should be treated!

<table>
<thead>
<tr>
<th>WARNING! WARNING! WARNING! WARNING! WARNING! WARNING!</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Frequent PVCs (greater than 6/min)</td>
</tr>
<tr>
<td>2. Runs of consecutive PVCs (couplets, triplets)</td>
</tr>
<tr>
<td>3. Multifocal PVCs</td>
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<tr>
<td>4. &quot;R on T&quot; phenomenon</td>
</tr>
<tr>
<td>5. Any PVC occurring in the setting of acute myocardial infarction</td>
</tr>
</tbody>
</table>
Case Study

While working in a cardiac step-down unit one night you are caring for a 79-year-old gentleman admitted with Atrial Fibrillation. Your patient is a heparin drip and an amiodarone drip. The patient also is showing signs of Congestive Heart Failure(CHF) and was started on 40 mg IV Lasix BID.

You noted the patient had the following rhythm run across the screen:

![ECG rhythm](image)

| Critical Thinking: What causes of PVCs might this patient have been experiencing? |

You make a call to the intern caring for this patient explained that the patient is in trigeminy. Although as an RN, you cannot prescribe, you do recognize this rhythm as a warning sign. The intern comes immediately to the room. The patient remained asleep and his blood pressure and heart rate remained within normal limits. His O₂ Sat was 98% on 2L O₂ via nasal cannula. Serum electrolytes including K⁺ and Mg²⁺ levels are drawn on the patient.

| Critical Thinking: You are investigating the possible electrolyte imbalances that might be causing this rhythm. What are other possible causes? |

Although an electrolyte imbalance is the likely cause, especially in a patient recently started on lasix, this patient has other risk factors for ventricular arrhythmias. The patient is taking amiodarone, which has the potential to cause arrhythmias. He also has signs of CHF that might indicate a dilated ventricle that can also be a cause of arrhythmias.

The intern leaves the room and the following rhythm runs across the screen. (Read the next section for an explanation of this rhythm strip!)
VENTRICULAR TACHYCARDIA

Three (3) or more consecutive PVCs characterize ventricular Tachycardia (VT). The conduction does not follow the normal pathway so the QRS complex is wide (greater than or equal to 0.10 sec). The QRS complex is normally followed by a large T wave in the opposite deflection of the QRS complex. The irritable foci have come from the ventricles and overtake the heart's normal pacemaker (SA node). The rate of VT is normally 100-250 beats/minute.

Ventricular Tachycardia can take on a few different shapes and sizes and the treatment may be different. Stop and take a closer look!

ECG Criteria
1. Heart Rate: 100-250 bpm
2. Rhythm: Regular ventricular rhythm
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: >0.10 seconds, wide and bizarre in appearance. It is difficult to differentiate between the QRS and the T wave. ST-T Waves are opposite deflection from QRS.
6. Rhythm Interpretation: Ventricular Tachycardia

Nursing Priorities
♦ Check your patient’s blood pressure to determine if this is stable, unstable (B/P <90) or pulseless VT.
♦ Treatment depends on the type of VT (stable, unstable, or pulseless.)
♦ Check your patient’s blood pressure, assess for syncope, SOB and chest pain.
♦ You patient may need to lie down to prevent potential falls.

Monomorphic VT

The rhythm is regular and the QRS complexes have a uniform appearance beat-to-beat. The first or the last beat may have a slightly different appearance.
Sustained VT is a VT that lasts more than 30 sec. or if less than 30 sec., requires intervention because of hemodynamic collapse (Thaler, 2003).

**Possible Causes**
- Myocardial irritability
- Acute MI
- Coronary Artery Disease (CAD)
- Drug toxicity
- Electrolyte imbalance
- Cardiomyopathy
- Idiopathic - of spontaneous and unknown origin

**Stop!** If this is your rhythm, you need to determine if this is stable or unstable VT.
- Assess your patient!
- ABCs (Airway, Breathing, Circulation)
- Vital signs

Is your patient symptomatic? Look for a decrease in level of consciousness, hypotension, shortness of breath, complaint of chest pain? If your patient is talking, oriented, has no complaints of chest pain or shortness of breath, and blood pressure is stable (>90 systolic blood pressure), then your patient is in Stable Ventricular Tachycardia.

**Treatment of Monomorphic Ventricular Tachycardia**

**Stable Ventricular Tachycardia**

Nursing interventions include:
- Administer Oxygen
- Page MD
- Get the emergency crash cart
- Make sure you have IV access
- Obtain a 12 lead ECG
- Stay with your patient and continue monitoring

Does your patient have a decrease in his/her level of consciousness? Is your patient complaining of shortness of breath or chest pain? Is there a drop in the blood pressure? Your patient is in a lethal arrhythmia! **Call a code!**

The physician may want to try an antidysrhythmic medication such as amiodarone, lidocaine, procainamide, or sotalol, first. If the medication is unsuccessful in converting this patient back to normal sinus rhythm (NSR), then you may need to cardiovert the patient. If the patient becomes unstable during the treatment, you MUST cardiovert the patient. ACLS has specific algorhythms for treatment of V Tach.
Unstable Ventricular Tachycardia

Nursing interventions include:

♦ Follow facility protocol
♦ Administer Oxygen (follow your facility protocol)
♦ Page MD
♦ Obtain IV access
♦ Consider sedation, if needed, with midazolam (Versed) or diazepam (Valium) (Cardioversion does hurt)
♦ Prepare to Cardiovert your patient

Synchronized Electrical Cardioversion

Every unit should be equipped with a monophasic or biphasic defibrillator or an automatic electrical defibrillator. Please see your facility’s policy and procedure manual on how to operate your particular defibrillator. DO NOT MEET YOUR DEFIBRILLATOR AT A CODE!!! Know how to operate the cardioversion, defibrillation, and the pacing mechanism of your facility’s defibrillator.

Cardioversion delivers an electrical shock on the “R” wave of the QRS complex. The defibrillator paddles are held in proper position on the chest and literally “watch” for an “R” wave. The shock is delivered on the “R” wave, thereby avoiding the vulnerable T wave. When preparing for cardioversion, be sure to select the lead with the tallest “R” wave.

Note: If the patient is defibrillated instead of cardioverted (synchronized), the patient may go into ventricular fibrillation. Ventricular fibrillation is caused when the electric current falls on the refractory period of the cardiac electrical cycle.

The exact procedures, number of joules, placement of electrodes and pads will depend on the type and possibly the brand of defibrillator used at your facility. Follow the specific guidelines for cardioversion keeping in mind that the device must be synchronized to the “R” wave in order to cardiovert the patient successfully.

If hypokalemia or hypomagnesemia are suspected as the cause, these can be treated concurrently with cardioversion. If your patient continues to be in Ventricular Tachycardia, await further orders from the physician. After the cardioversion, it is possible that your patient has returned to NSR. It is also possible that your patient's condition has deteriorated and now the patient is in Ventricular Fibrillation (VF) or Asystole. We will discuss these conditions later in this course.

Early cardioversion is the best treatment you can give to your patient with VT. Amiodarone is generally then started to deter further arrhythmias.
Polymorphic Ventricular Tachycardia

What is happening here? This does not look like the previous strip of VT. Polymorphic V Tach indicates that the patient has more than one irritable focus in the ventricles. The QRS complexes are wide with varying shapes.

Stop! If this is your rhythm, you need to determine if this is stable or unstable VT.

♦ Assess your patient!
♦ ABC's (Airway, Breathing, Circulation)
♦ Vital signs

ECG Criteria
1. Heart Rate: 100-250 bpm
2. Rhythm: Irregular
3. P waves: May or may not be seen
4. PR Interval: Not applicable
5. QRS Width: > 0.10, wide and bizarre, differ one from another. ST-T Waves: Differ with every beat, may or may not be seen.
6. Rhythm Interpretation: Polymorphic Ventricular Tachycardia

Nursing Priorities
Check your patient’s blood pressure. Can you find a pulse? Is the patient stable or unstable? If you cannot find a pulse, go to the treatment of VF (discussed later in the course). Pulseless VT is treated as VF.

Treatment of Polymorphic Ventricular Tachycardia
If the Polymorphic VT does not have a prolonged QT interval, it is treated with amiodarone or other antidysrhythmic drugs and cardioversion, as in the previous section on monomorphic VT.
Torsades De Pointes

However, there is a specific type of Ventricular Tachycardia that does have a prolonged QT interval. This lethal rhythm is called Torsades de Pointes. Torsades de Pointes is French for "twisting of the points." Let's look at a strip.

![ECG Strip](image)

See how the QRS complexes appear to twist in a spiral around the baseline? The rhythm is precipitated by a prolonged QT interval. In the sinus beat preceding the event, the QT interval is $\geq 0.50$ sec. A PVC falls on the prolonged T wave and sets off the Torsades de Pointes. You will see a short pause and then the QRS complexes will peak up then down. The rate is normally very rapid (200 - 250 beat/min.). The rhythm is usually poorly tolerated. This rapid rate leads to extremely diminished cardiac output. Seizures and syncope are common with this dysrhythmia. If not stopped quickly, this rhythm will be lethal.

**ECG Criteria**
1. Heart Rate: 200-250 bpm
2. Rhythm: Irregular
3. P waves: Not seen
4. PR Interval: Not applicable
5. QRS Width: $> 0.10$, QRS complexes are wide and bizarre, differing in size and deflection. ST-T Waves: Not seen
6. Rhythm Interpretation: Torsades de Pointes

**Nursing Priorities**
- Assess your patient.
- Have the defibrillator nearby, obtain IV access.

**Possible Causes**
- Medications that prolong the QT interval: Quinidine, procainamide, disopyramide, sotalol, amiodarone, propafenone
- Electrolyte imbalances
- Severe bradycardias
- Liquid protein diets
- Mitral valve prolapse
- Central nervous system lesions
**Prevention**

Monitor the QT interval when your patient is on one of the medications that cause a prolonged QT interval. If the QT interval reaches 0.56 - 0.60 sec., notify the physician. The physician will need to modify the dosage of the medication.

**Treatment of Torsades De Pointes**

- Sometimes this rhythm stops spontaneously.
  
  Magnesium is the drug of choice for Torsades de Pointes. Magnesium Chloride or Magnesium Sulfate may be given IV push followed by an infusion over several hours.

- Override ventricular pacing may be attempted.

- If all of these interventions fail, cardioversion will be necessary. Follow the above guidelines for cardioversion.

**General Treatment of Ventricular Tachycardia**

The American Heart Association in Collaboration with the International Liaison Committee on Resuscitation put out the *Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: An International Consensus on Science* in August of 2000. The new Advanced Cardiac Life Support guidelines now list specific algorithms for monomorphic VT, polymorphic VT, and Torsades de Pointes. The specific guidelines should be available at your facility guidelines and through the American Heart Association (AHA) Web Site or at www.acls.net.
Case Study

Mr. Smith is a 74 year-old gentleman that was admitted to the hospital with complaints of chest pain and shortness of breath. He was given Aspirin 325mg po in the ED upon admission. He was started on a Nitroglycerin drip at 25mcg/min. He has now been transferred to your telemetry floor. His admitting diagnosis is Rule Out MI. You take an initial set of vital signs: BP 118/58, pulse 90 irregular, respiration rate 22, O2 Sat 94% on 4L O2 NC. He states he is now no longer experiencing chest pain but has a terrible headache. Before leaving the room, you place Mr. Smith on a telemetry monitor. You hand him the call bell and assure him you will be back shortly with Tylenol. You walk out to the desk and glance at the bank of telemetry monitors. You are not surprised to see this rhythm for Mr. Smith.

What else would you like to know?

Mr. Smith’s Medical History:
♦ Past medical history: Previous Inferior Wall MI two years ago, Percutaneous Transluminal Coronary Angioplasty to the Right Coronary Artery two years ago, Hypertension, Congestive Heart Failure, and Non Insulin Dependent Diabetes Mellitus.
♦ Past surgical history: Right total hip replacement four years ago
♦ Current medications:
  ♦ Aspirin 325 mg po Daily
  ♦ Atenolol 25 mg po BID
  ♦ Zestril 5 mg po Daily
  ♦ Lasix 40 mg po Daily
  ♦ KCL 20 mEq po Daily
  ♦ Digoxin 0.125mg po Daily
  ♦ Glucophage 500 mg po BID
  ♦ Lasix 40 mg po Daily
  ♦ Extra Strength Tylenol 1000mg po q4-6 hrs PRN for pain
♦ Admission Labs:
  ♦ WBC 6.7
  ♦ Hgb 10.8
  ♦ Het 30.7
  ♦ Plt 186
  ♦ Mg++ 1.6
  ♦ Ca++ 7.9
  ♦ Na+137
  ♦ BUN 24
  ♦ K+ 3.5
  ♦ Creat 1.0
  ♦ CPK 509
  ♦ CKMB 34.1
  ♦ Troponin 2.6
♦ Admission ECG: Normal sinus rhythm with frequent PVCs, Q waves present in leads II, III, and avF.

You are standing at the med cart when you hear a nurse call for help. You turn to see the nurse standing in the doorway of Mr. Smith's room. The nurse tells you Mr. Smith is difficult to arouse. You grab the emergency crash cart and head for the room. After connecting the telemetry leads to the defibrillator monitor, you see this rhythm.
Mr. Smith’s blood pressure is 86/40. He has a faint, palpable pulse. The rate is difficult to count, but you believe it to be around 160. He is diaphoretic and respiration rate is 32. His O2 Sat is now only 86% on 4L O2 nasal cannula. You grab the 100% non-rebreather mask from the crash cart and place him on 10L O2. Another nurse calls a code. You ask her to stay at the head of the bed and monitor his airway. The secretary pages Dr. Brown for you.

**Critical Thinking:** What rhythm do you think you are seeing? How can you verify your initial thoughts? How does your patient assessment fit in with your initial interpretation, or does it make you think there may be another option? What might have caused this condition? What are your treatment options?

Your patient has a fast regular rhythm with QRS complexes that are wider than 12mm. Your initial interpretation is V Tach. The complexes all look the same, so you further define the rhythm as monomorphic VT. Your patient vital signs have deteriorated, so the VT is unstable. His labs show that his potassium and calcium are low and his magnesium is borderline low. In addition, Mr. Smith has a history of coronary artery disease and his hospital course is consistent with acute myocardial ischemia. What should you do next? Prepare for cardioversion!

The intern caring for this patient arrives first to the scene and orders the Nitroglycerin drip to be shut off. (This can also cause hypotension.) The intern asks for a bag of .9% NS to be hung. (Having a bag of IV fluid running during a code is always a good idea!) You have already placed the gel conductor pads on the patient's chest in the appropriate spots. The intern grabs the defibrillator paddles, places them on the patient's chest and asks you to charge the defibrillator to 100joules. "I'm going to shock on 3. One, I'm clear. Two, you're clear." (You back away from the bed.) "Three, we're all clear." The intern discharges the paddles. You realize too late that the “synchronize” button was not selected, so the defibrillator did not wait to see the “R” wave before discharging the current. You now see this rhythm.

**Critical Thinking:** What happened? What may have caused this rhythm?

Defibrillating VT instead of cardioverting (synchronized defibrillation) can cause the electric current to fall on the refractory period of the cardiac electrical cycle. This is a vulnerable time in the electric cycle. The patient may go into ventricular fibrillation, as this patient did.
VENTRICULAR FIBRILLATION

Ventricular Fibrillation (VF) is the result of chaotic electrical activity in the ventricles from either repetitive small areas of re-entry or from a series of rapid discharges from various irritable foci within the ventricular myocardium. This chaos causes the ventricles to quiver. Scientists who have visualized the ventricular myocardium during VF describe it as “a can of worms.” When the ventricles quiver, there is no cardiac output. You will not be able to hear any audible heart tones. There will be no palpable pulse. There will be no blood pressure. The patient will become cyanotic. Apnea and seizures are common. Clinically, you will be unable to differentiate VF from Asystole (covered next). Without quick intervention, the rhythm will be lethal within 3-5 minutes!

The ECG appears as a continuous, undulating pattern without clear P waves, QRS complexes or T waves (Thaler, 2003).

![ECG pattern](image)

**ECG Criteria:**

1. Heart Rate: not able to be determined
2. Rhythm: Irregular
3. P waves: Not seen
4. PR Interval: None
5. QRS Width: Fibrillatory waves; cannot determine. ST-T Waves: Not seen.
6. Rhythm Interpretation: Ventricular Fibrillation

**Nursing Priorities**

- Check rhythm in a second lead.
- Assess your patient for ABCs
- If confirmed, call a code!

**Coarse VF**

Coarse VF is large, erratic undulations around the baseline. This usually indicates new onset of VF and is usually more easily defibrillated.

**Fine VF**

Fine VF is gentle undulations around the baseline. It resembles a tremor. Fine VF usually indicates that the patient has been in this rhythm for a short time. Medications can be given to try and change fine VF into coarse VF to make the rhythm more refractory to defibrillation. Epinephrine is the drug of choice for VF.
Sudden death from cardiac causes, often due to ventricular fibrillation, claims at least 340,000 persons annually in the United States. (American Heart Association, 2005).

Possible Causes:
- Acute MI
- Untreated Ventricular Tachycardia
- Cardiomyopathy
- CAD
- Acid base imbalance
- Electrolyte imbalances

Treatment of Ventricular Fibrillation:
Remember you only have 3-5 minutes before your patient dies!
- What are you going to do? Defibrillate!
- What was that? Defibrillate!
- One more time! Defibrillate!

“DON’T WAIT, DEFIBRILLATE!”

That's right! Early defibrillation is the key to survival for a patient in Ventricular Fibrillation.

Precordial Thump – What is Your Facility’s Policy?
A precordial thump may be helpful if the cardiac arrest is witnessed. If the patient does not have a pulse and the defibrillator is still on the way, you may perform a forceful precordial thump. Raise your arm above the patient’s chest, lower your forearm with your fist closed and thump the patient on the chest over the heart. It just may convert your patient out of V-fib or pulseless VT. Caution: If your patient did have a coordinated rhythm and you perform a precordial thump, you can also convert them into VF or Asystole (Cummins, 1997).
Let’s return to Mr. Smith. Where were you? You are standing next to the bed and you see this rhythm across the monitor.

The rhythm above is coarse VF. Defibrillation is the treatment of choice and is generally performed in 3 "stacked" shocks. If the patient is converted out of the rhythm by the first one or two, you obviously stop. Otherwise, perform the 3 consecutive shocks. (In addition to managing this patient’s rhythm, don’t forget that airway management if the “first” of the ABC’s of CPR.)

Epinephrine is generally the first drug of choice with VF. The usual dose is 1 mg IV. It can be repeated every 3-5 min. If the VF still persists, you can consider an antiarrhythmic drug. Amiodarone is now the first choice of IV drugs used in this situation (ACLS.net, 2005).

The physician performs 2 consecutive stacked shocks. Fortunately you now see this rhythm on the monitor screen.

Your patient has a pulse! Congratulations! Mr. Smith is then intubated and his blood pressure is 70/36. He is being transported to the intensive care unit.

**Remember**: As soon as VF is detected, establish an airway, begin CPR, and obtain IV access.

**Ventricular Fibrillation Mnemonic**

ACLS has put out the following mnemonic to help healthcare providers remember the order of running a VF code. Check with your institution before applying these guidelines as practices do differ.
These are guidelines!

Please - **P**recordial **T**hump  
**S**hock - 200 joules  
**S**hock - 300 joules  
**S**hock - 360 joules  
Everybody - **E**pinephrine 1 mg IV q 3-5 min. or e**V**erybody - **V**asopressin 40 u IV  
**S**hock - 360 joules  
And - **A**miodarone 300 mg IV push, may repeat once at 150 mg in 3-5 min.  
Let's - **L**idocaine 1.0-1.5 mg/kg IV. May repeat in 3-5 min.  
Make - **M**agnesium Sulfate 1-2 g IV (2 min. push)  
**P**atients - **P**rocainamide 20 mg min or 100 mg IV q 5 min.  
Better - **B**icarbonate 1mEq/kg IV  
(Cummins, 2000; ACLS.net, 2005)

As previously stated, the new ACLS guidelines came out in August, 2000. The new changes for VF and VT include using only one antiarrhythmic drug per patient and using IV Amiodarone as a first choice drug over Lidocaine. Another change was adding Vasopressin as the recommended alternative to Epinephrine. Magnesium is now only recommended in known hypomagnesemia and Torsades de Pointes. Bretylium has "been removed from ACLS treatment algorithms and guidelines because of the high incidence of adverse reactions and the availability of safer drugs" (Asselin, 2001).

**Long Term Treatment Options for Ventricular Arrhythmias**

**Electrophysiology study**

**EPS** is a study performed where a catheter is inserted into the right side of the heart under fluoroscopic guidance, much like a cardiac catheterization. The catheter detects the electrical impulses of the heart. Is the electrical impulse following the normal pathway? Are any arrhythmias noted? The physician can also use the pacing capacity to try and create an arrhythmia. The physician is trying to locate the irritable focus (foci) that may have caused the VT of VF in your patient. If the irritable focus is found, radio frequency ablation of the source of the arrhythmia may be used.

**Radiofrequency Ablation and Automatic Implantable Cardioverter Defibrillators**

**Radiofrequency Ablation** is where a catheter is used to send low voltage, high frequency alternating current to the tissue to cauterize the area. If the irritable focus is not found, an **Automatic Implantable Cardioverter Defibrillator** (AICD or ICD) can be placed. This procedure takes place in the cardiac catheterization lab or in the operating room. The small generator is placed in a pocket of muscle under the skin, once in the abdomen and now more frequently, below the left clavicle. Sensing and defibrillation leads are attached to the generator and threaded into the heart or the tissue surrounding the heart. The leads can sense an arrhythmia and send the information to the generator, and the generator can send an electric current (small amount of joules) through the leads to defibrillate the heart. The physician can program the ICD with a set number of joules to be used and how many beats of VT or seconds of VF to allow before defibrillating the patient. Example: 26 beats of VT, defibrillate with 6 joules. A much smaller amount of joules can be used because the leads are resting on or in the heart. The current does not have to pass through skin, muscle, or bone before reaching the myocardium. The patient will still detect the shock but often remains conscious. The patients will also be on antiarrhythmic drugs. All of the efforts are in hopes of preventing or quickly correcting a second lethal arrhythmia.
What’s the Difference in the new Defibrillators?”

♦ Monophasic Defibrillator

Current flows in one direction from one paddle/pad to the other in a monophasic defibrillator. In VF or Pulseless VT, 3 “stacked” shocks are recommended to be delivered consecutively in the order of 200 joules, 300 joules, then 360 joules.

♦ Biphasic Defibrillator

In a biphasic defibrillator electrical current flows in one direction during the first phase and reverses direction in the second phase (thus passing through the heart twice). Biphasic defibrillators are able to utilize fewer joules and produce less myocardial damage with equal to or better results than monophasic defibrillators. The biphasic technology is being used in Automatic Internal Cardiac Defibrillators (AICDs) and Automated External Defibrillators (AEDs). Research is still being done on the biphasic defibrillators and no recommendation has yet been made by the American Heart Association in terms of the level of joules to be used during VF or Pulseless VT (American Heart Association, 1998). Much of the literature suggests that for VF/PVT 150 joules, 150 joules, 150 joules be used for the three consecutive shocks.

♦ Automated External Defibrillator (AED)

An AED is a biphasic defibrillator that uses a “hands off” approach. The operator does not have to interpret the patient’s heart rhythm; the defibrillator does this for you. The defibrillator will also choose the appropriate action needed for that particular rhythm. The AED consists of a small portable unit with a set of defibrillation electrode pads. When energized, a voice will prompt the operator what to do next. The pads come with pictures to instruct on placement. Once the pads are placed on the patient, the AED will read and interpret the patient’s rhythm. Next, the voice will prompt you to “shock” the patient, if needed. The operator needs only to push the “shock” button. The AED will determine the joules. The AED does not have adjustable settings. Besides the defibrillation electrode pads, the features on an AED consist only of an “on/off” button, monitor screen, and a “shock” button. AEDs are now being placed in “crowded” areas all across the world such as airports, stadiums, shopping malls, and airplanes. The new ACLS guidelines state “All healthcare providers who are expected to perform cardiopulmonary resuscitation should be trained in the use of automated external defibrillators and authorized to use them.” (Asselin, 2001, p. 49) The goal of AEDs is to reduce the amount of time between when the patient goes into VF and the time of defibrillation.
**ASYSTOLE**

Asystole is seen as a flat line across the monitor screen. Asystole is the absence of any electrical activity. Clinically, your patient will be unconscious, apneic and pulseless. As mentioned before, it is difficult to differentiate between Asystole and VF without a telemetry reading. However, the treatment is very different. Always verify this rhythm in two leads.

**ECG Criteria**

1. Heart Rate: None
2. Rhythm: None
3. P waves: None
4. PR Interval: None
5. QRS Width: None; ST-T Wave: None
6. Rhythm Interpretation: Aystole

**Nursing Priorities**

Check rhythm in a second lead. If your patient has a pulse, they are obviously NOT in asystole. If your patient is pulseless initiate CPR and call for help!

**Possible Causes**

- Severe metabolic deficit
- Acute respiratory failure
- Extensive myocardial damage or ruptured ventricular aneurysm
Defibrillation does not "jump start" the heart. Defibrillation actually stops the electrical activity. This allows for the heart's own pacemaker (SA node) to take over.

**Case Study**

Mrs. Wilson is a 90 year-old woman admitted with sepsis. She is on 100% non-rebreather mask with an O2 sat of 94%. Her respiration rate is 30, HR 56, BP 96/50, rectal temp 96.0°F. She is lethargic and disoriented to place and time. She has a past medical history of chronic renal failure, diabetes mellitus peripheral vascular disease and hearing loss in one ear. She is scheduled for dialysis later today. Blood Cultures x2 are pending. A urinalysis with culture & sensitivity have been sent. A chest X-ray revealed bilateral pleural effusions. You are in the middle of shift change and are giving report on this patient when . . .

The telemetry technician on your unit shouts your name and tells you go to into room 344 (Mrs. Wilson’s room). The monitor above her bed shows…

STOP! Assess the patient. She may have just pulled a lead off.

Mr. Wilson is cool to touch and is not arousable. You shake her and shout into her "good" ear. She is unresponsive. All her leads are in place. You shake her again with no response. You open her airway and determine she is not breathing. You grab the mask above the bed and give her two breaths. You search for a pulse and do not find one. You call for help. A check in a second lead confirms asystole. What will you do next? Do you want to prepare for defibrillation? No!

**Treatment of Asystole**

Treatment of asystole is through the use of CPR and IV meds until some kind of rhythm is established. Epinephrine followed by atropine is generally used. The goal is to “irritate” the heart into some kind of rhythm that then can be “shocked.

Transcutaneous pacing is now one of the standard treatments for asystole.
Transcutaneous Pacing

Transcutaneous pacing can be used to stimulate cardiac activity during a code. During transcutaneous pacing, an electrical current is passed through an electrode placed on the skin of the chest to stimulate electrical activity in the heart. There are other means of “pacing” the heart available, but this approach is the quickest and most readily available in a crisis situation. Most defibrillators on your units will have a pacing capability built into them. If not, a separate transcutaneous pacemaker will most likely be available during a code situation. Learn about the pacing capabilities on your floor prior to a code.

♦ Regardless of the brand of transcutaneous pacer used, there are several key facts to keep in mind when attempting transcutaneous pacing:

♦ If your patient is awake or aware, sedation may be necessary as the procedure may be uncomfortable

♦ Clean and dry skin is necessary under each of the “pads” used to pace in order to assure adequate conduction.

♦ The demand mode of pacing is used most often. If the demand mode is used, the pacer only stimulates the heart if the rate falls below a certain preset point.

♦ The QRS will look wide and bizarre as it is being stimulated outside of the normal pathway.

♦ An adequate ECG is not assurance that the pacer is helping the patient. Assess your patient to be certain that the electrical conduction is followed by the mechanical action of the heart, thereby causing a pulse and blood pressure.
ADDITIONAL ARRHYTHMIAS

Pulseless Electrical Activity

Pulseless Electrical Activity (PEA) is a cardiac rhythm where there is the presence of electrical activity but no detectable pulse. Several rhythms fit into this category, what they all have in common is that there is no cardiac output associated with the rhythm you see on the screen. These rhythms include: Electromechanical dissociation (EMD), pseudo-EMD, idioventricular rhythms, ventricular escape rhythms, post defibrillation idioventricular rhythms, and bradyasystole rhythms. Your patient will be unconscious, apneic, and pulseless. The most common cause of PEA is hypovolemia.

ECG Criteria
Depend on the particular underlying rhythm.

Nursing Priorities
Assess your patient. Check airway, breathing, and circulation. Through your assessment you may determine the cause of PEA. Treat the cause!!!

Treatment of PEA
The treatment of PEA depends on the cause, so determining the cause is critical. Common causes of PEA include:

- Hypovolemia
- Hypoxia
- Hypothermia
- Hyper-/hypokalemia
- Hydrogen ion – acidosis
- Tamponade, Cardiac
- Tension pneumothorax
- "Tablets"- drug overdose
- Thrombosis, coronary (acute, massive MI)
- Thrombosis, pulmonary (massive PE)
Agonal Rhythm

Agonal Rhythm is a slow, irregular rhythm with wide QRS complexes of varying morphology. This rhythm is seen in the late stages of unsuccessful resuscitation attempts. The myocardial tissue is dying. The QRS complexes become wider and wider and eventually the rhythm ends in asystole. The patient is unconscious, apneic, and pulseless.

ECG Criteria
1. Heart Rate: ≤20 bpm
2. Rhythm: Irregular
3. P waves: None
4. PR Interval: None
5. QRS Width: >0.10 seconds wide and bizarre, becomes more and more prolonged. ST-T Wave: May or may not be seen, if seen, very wide
6. Rhythm Interpretation: Agonal rhythm

Nursing Priorities
An agonal rhythm is generally seen at the end of an unsuccessful code or when a patient has a “Do Not Resuscitate” order. Nursing priorities generally are:
♦ Consider termination of efforts.
♦ Family support may be needed
CONCLUSION


The following case history describes the first instance of lifesaving defibrillation performed by a nurse in the absence of a physician. This event took place in 1963 and became the precedent for the now established practice of defibrillation by nurses.

A 72-year-old male was admitted to the CCU of the Presbyterian-University of Pennsylvania Medical Center with a history of chest pain that had subsided by the time of his arrival. He had no complaints and in fact, wanted to go home. An ECG showed an AMI. The findings upon physical examination were normal and there was no evidence of complications. The patient remained in normal sinus rhythm, with a heart rate ranging from 60-74/minute. Occasional premature ventricular beats were noted.

Some 60 hours after admission, in the middle of the night, the patient’s cardiac monitor alarm sounded. The nurse instantly recognized VF on the monitor and ran to the bedside, where she found the patient to be unconscious. The nurse immediately called the physician and set a timing device for 2 minutes. She turned on the defibrillator, set the energy level at 200joules, and applied electrode paste to the defibrillator paddles. The nurse defibrillated the patient without further delay. Normal sinus rhythm was established almost immediately. The patient survived and was still alive 10 years later.”

As you can see by the story above, lethal arrhythmias can occur at anytime, and rapid nursing assessment and intervention can result in a successful outcome. Quick recognition of lethal arrhythmias and strong working knowledge of up-to-date treatment will lead to the best outcome possible.
REFERENCES


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