**Prehospital 12-Lead ECG**

**Objectives**

At the end of the lesson the student should be able to:

1. Discuss cardiac anatomy as it relates to 12-lead electrocardiogram (ECG).
2. Recognize the need for 12-lead acquisition and transmission and describe proper lead placement.
3. Identify 12 lead changes indicating acute coronary syndrome (ACS).
4. Describe the management of acute coronary syndrome patients.

**Case Study**

The crew of Medic 14, a double paramedic crew in an urban EMS system, just sat down to lunch at their favorite spot when the radio crackles and dispatch ruins their cheeseburger pipe dreams. Dispatch reports a male in his fifties with chest pain and difficulty breathing on the 53rd floor cafeteria of a nearby office building.

Upon arrival on scene the crew finds a middle aged overweight male sitting at a table in the cafeteria. The patient is conscious, alert and fidgeting anxiously. He is pale, diaphoretic, and his right hand is balled into a fist and is pressed against the left side of his chest. The patient complains of severe retro-sternal chest pain radiating to his left arm and jaw. The patient describes the pain as heaviness like someone is sitting on his chest. The pain began suddenly while walking from his office to the cafeteria. The pain is rated 10 out of 10. The patient also complains of shortness of breath and nausea. He vomited once prior to the crew’s arrival. During the assessment, the patient states that he feels like he is about to die.

The patient’s initial vital signs are a heart rate of 90 beats per minute strong and regular radially, a blood pressure of 170/100 mmHg, respirations of 20 breaths per minute and labored, lung sounds that are clear and equal in all fields, and an SPO2 of 97% on room air. The patient has a history of hypertension and elevated cholesterol. He denies allergies and reports taking daily lisinopril and Zocor. The patient further denies the use of Viagra or other medications for erectile dysfunction.

The crew immediately places the patient on high flow oxygen via non-rebreather mask, applies their cardiac monitor and obtains a 12-lead electrocardiogram. The ECG shows marked ST segment elevation in leads II, III, and AVF with reciprocal T wave inversion in lead I. This is strongly indicative of an inferior wall myocardial infarction. This typically results from an occlusion of the right coronary artery. Once the ECG was obtained, one member of the crew transmitted the information to the receiving facility which is capable of emergent percutaneous coronary intervention and also contacted online medical command in order to mobilize the catheterization lab team as soon as possible.

The other member of the crew, administers 324 mg aspirin orally, 1 spray of sublingual nitroglycerin and establishes IV access. The patient reports that his pain continues to be a 10 out of 10 after the medication is administered. The patient’s vitals are monitored closely and rapid transport is initiated.

While en route to the hospital, the crew monitors the patient’s ECG for changes. Two more doses of sublingual nitroglycerin are administered with no relief of the patient’s pain. The patient’s blood pressure remains stable at 150/90 mmHg after the nitroglycerin. The crew administers 4 mg of morphine sulfate intravenous push (IVP). The patient also continues to complain of nausea. Therefore, 4 mg of Zofran (ondasteron) IVP is administered. After the morphine and Zofran, the patient’s pain is now a 5 out of 10 and his nausea is relieved. Finally, a secondary IV access is established. The patient is much calmer and rests comfortably on the stretcher.

Upon arrival at the hospital, the crew is met at the door by the attending physician. She quickly assesses the patient while he is still on the stretcher. The cath lab team is ready and standing by thanks to the early notification provided by the EMS crew. The emergency department is bypassed and the crew takes the patient directly to the cath lab where an emergency angioplasty is performed.

It is later determined that the patient had a 90% blockage of the right coronary artery that was successfully stented. Cardiac reperfusion occurred within 30 minutes of arrival at the hospital and the damage to the myocardium was minimal. After a three-day hospital admission the patient was released in good condition.

**Introduction**

The prehospital 12-lead electrocardiogram (ECG) is one of the most important diagnostics in the paramedic’s scope of practice. The ECG is the most accurate and sensitive indicator of ST segment elevation myocardial infarction (STEMI) in patients with symptoms of acute coronary syndrome (ACS). According to the Centers for Disease Control, acute myocardial infarction (MI) is a leading cause of death in the USA. Rapid treatment and transport of the acute MI was one of the inspiring goals of paramedicine at its inception. Since its birth in the late 1960’s, prehospital treatment of the ACS patient has grown significantly. The technology that is available today brings unprecedented levels of care directly to the patient. The cardiac monitors that are currently in use may allow for the immediate transmission of 12-lead ECGs, and are becoming the standard of care in most parts of the country. Early reperfusion that is facilitated by field identification of a potential infarction is a central factor in the successful treatment of the STEMI.

Studies show that 911 to balloon time can be minimized if STEMI patients are identified in the field and triaged to the appropriate facility. Current research shows that the triage of ACS patients to facilities that are capable of performing emergency percutaneous cardiac intervention (PCI) improve patient outcomes just as the triage and transport of trauma patients to trauma centers does. In order to obtain a diagnostic 12-lead ECG and treat the ACS patient appropriately, the paramedic must have a thorough understanding of cardiac anatomy and the symptom presentation. The paramedic must also know the proper placement of the ECG leads,
recognize the changes in the ECG that indicate myocardial infarction, and be well versed in current treatment options.

**Cardiac Anatomy**

The heart is the major organ of the circulatory system and is responsible for blood flow throughout the entire body. It can be described as a two stage pump that is at the nexus of the pulmonary and systemic circulation. The heart muscle is unique due to its automaticity at the cellular level. The cells within the myocardium of the heart are the only cells in the body that have automaticity or the ability to generate their own electrical current. This generation of electrical current is what enables the heart to beat without the individual continually thinking about it. Imagine what it would be like if you had to tell your heart to beat just like you have to think to walk.

The heart is located in the retrosternal area between the lungs also known as the mediastinum and is approximately the size of a closed fist. Essentially the heart is a specialized muscle. The muscle tissue of the heart is known as the myocardium. The heart is contained within a thick fibrous membrane that encloses it like a sac known as the pericardium. The pericardium functions to protect the heart from external infection and injury. It is also anchored to the chest wall and works to hold the heart in place. If there is damage to the pericardium and bleeding occurs, it can cause a life-threatening condition known as pericardial tamponade. This develops as blood builds up within the pericardium, and puts pressure on the heart, impeding the heart’s ability to fill and contract properly.

The heart is divided into four chambers. There are two atria and two ventricles. Those chambers are divided along a vertical axis and are categorized as the right side and left side. They are separated by septums known as the interatrial septum and the interventricular septum. To better understand the structures of the heart, we will now follow a drop of deoxygenated blood from the systemic circulation as it makes its way through the cardiac cycle. The deoxygenated blood flows into the right atrium from the systemic circulation via the inferior and superior vena cava as well as the coronary sinus. The coronary sinus collects the blood used in the heart muscles. Once in the right atrium, the blood is pushed through the tricuspid valve into the right ventricle. From the right ventricle, the blood flows through the pulmonary semilunar valve into the pulmonary arteries. Once in the pulmonary circulation, the blood moves into the lungs. In the lungs, carbon dioxide is released and oxygen is bound to the red blood cells. Now that the blood has become oxygenated it flows back to the heart through the pulmonary veins. The oxygenated blood flows into the left atrium and then is pushed through the bicuspid valve into the left ventricle. From there it passes through the aortic semilunar valve into the aorta. The oxygenated blood then perfuses the rest of the body as well as the heart itself. It is important to note that the many valves of the heart keep the blood from flowing backwards. If a valve fails it can lead to significant complications.

Once the basic anatomy and function of the heart is detailed, the perfusion of the heart must be considered. The myocardium requires oxygen and nutrients just like every other muscle in the body. The coronary arteries which branch off from the aorta right above the left ventricle supply the heart. The coronary arteries are subdivided into a right coronary artery (RCA) and a left coronary artery (LCA). The RCA is further divides into nine branches: the conus branch, sinus node branch, right ventricular branch, atrial branch, acute marginal branch, atrioventricular node branch, posterior descending branch, left ventricular branch, and left atrial branch. These branches supply oxygenated blood to the right atrium and right ventricle with a small amount also going to the inferior part of the left ventricle and the cardiac conduction system. The LCA is divided into two main branches known as the left anterior descending artery and the circumflex coronary artery. These vessels supply the left side of the heart and the intraventricular septum. Knowledge of this system is integral in the discussion of coronary artery disease and STEMI because it is these vessels that become occluded causing infarction of the myocardium.

In order to further facilitate the following discussion of electrocardiograms and 12-lead ECGs the electrophysiology of the heart must also be understood. The mechanical pumping action of the heart is driven by chemical changes that occur in the myocardium at the cellular level. All the cells in the heart have the automaticity that was previously mentioned. Under normal circumstances, however, there are standard conduction pathways that are utilized. This system consists of six basic parts: the sinoatrial node (SA node), the atrioventricular node (AV node), the bundle of His, the right and left bundle branches, and the Purkinje fibers.

The cardiac conduction cycle normally arises high in the right atrium at the sinoatrial node. In the healthy heart this is the primary pacemaker and generates 60 to 100 beats per minute. The impulses generated in the sinoatrial node cascade through the right and left atrium causing cellular depolarization and contraction of the atria. This produces the P waves on the standard ECG. There is then a pause as the depolarization cascade arrives at the atrioventricular node. This node functions like an electrical capacitor where the charge gains strength prior to spreading into the bundle of His. From there the charge spreads throughout the ventricles via the bundle branches and the Purkinje fibers. This pause at the atrioventricular node is seen on the ECG as the brief return to the isoelectric line between the P wave and the QRS complex.

In order for this cascade of electricity to move through the conduction system, depolarization must occur at the cellular level. This happens because each individual cell has a relatively negative ionic charge as compared to the interstitial space. The fact that the inside of the cells are more negatively charged than the outside creates a resting action potential across the cell membrane. Therefore when stimulation occurs, there is a flood of positive ions down the electrical gradient across the cellular membrane into the cell. The primary ions that are responsible for this process are sodium, potassium, and calcium. That is why electrolyte problems can have such a profound effect on the cardiovascular system and why calcium channel blockers are used to slow down rapid
dyssrhythmias. When a cell in the myocardium receives a stimulus, sodium and calcium rush into the cell, moving along the electrochemical gradient. The movement of ions is what creates the electrical current and continues until the charge within the cell becomes slightly positive. This is depolarization which stimulates the release of calcium into the muscle fibers stimulating muscular contraction. Once depolarization has occurred, the cells must return to their resting state though a process called repolarization. First, potassium passively flows out of the cell. Then, the sodium is actively pumped out via the sodium potassium pump. Finally, the calcium is reabsorbed. The process of repolarization has two phases. First is the absolute refractory period in which the cell cannot respond to any electrical stimulation. The second is the relative refractory period in which the cell has not yet returned to its full resting potential but a strong stimulus can initiate depolarization. The ECG is a visual measurement of this depolarization and repolarization.

This complex electrical system is regulated primarily by the autonomic nervous and the endocrine systems; however the individual has some ability to control heart function. The overall heart function is controlled by three regulatory mechanisms of the autonomic nervous system. The first is the chronotropic state which is the rate of contraction. The second is the dromotropic state which is the rate of electrical conduction through the myocardium. Finally, the third is the inotropic state, which is the strength of each contraction of the heart. The heart muscle itself also influences the strength of contraction and cardiac output. The Frank-Starling mechanism states that, like a rubber band, with increased stretching the heart muscle contracts with greater force to a limit. This means that with increased force of contraction there is an increase in output.

The nervous system has chemoreceptors which sense chemical changes in the blood, and baroreceptors which sense changes in blood pressure. If any of these receptors are stimulated, hormones or neurotransmitters are released as the body attempts to return the system to its normal state of homeostasis. Those chemical and neurological changes are functions of both the sympathetic and parasympathetic nervous systems and can affect heart rate and contractility. Parasympathetic stimulation, relating to rest and digest functions, slows the heart and sympathetic stimulation, the fight or flight mechanism, increases the heart rate and contractility.

**Indications for 12-lead Acquisition and Transmission**

The 12 lead ECG is a diagnostic that can provide vital information. Any patient who presents with symptoms of acute coronary syndrome should be evaluated with this tool. The American Heart Association defines acute coronary syndrome as any condition that involves blocked or decreased blood to the heart muscle with symptoms that include: chest pain or discomfort that may involve pressure; tightness or fullness; pain or discomfort in one or both arms, the jaw, neck, back or stomach; dyspnea; feeling dizzy or lightheaded; nausea; and diaphoresis. Body language is important, too. Levine’s sign, when a patient’s description of chest pain includes a closed fist placed in the center of the chest, is thought to indicate cardiac ischemia 77% of the time. These are the classical presentations of ACS, but you should always be on the lookout for patients that present atypically. Patient populations such as elderly women, diabetics, and heart transplant patients are much more likely to have myocardial infarctions without chest pain. In fact, 10 or 20% of all AMI patients do not experience any chest pain at all. These chest pain free patients may complain of sudden unexplainable weakness, loss of appetite, persistent hiccups (caused by irritation of the diaphragm by inferior wall MI), nausea, sweating, and anxiety, or a feeling of impending doom. The bottom line is that any patient who is at risk for cardiovascular disease and has a complaint that is not obviously related to another existing condition should receive a 12-lead ECG. No one will ever fault you for performing ECGs that come back normal. However, if EMS delivers a patient to the emergency room who ends up having an active STEMI that went undiagnosed in the field, there will likely be consequences for those providers.

While you should be very cautious to rule out acute coronary syndrome in the field without performing a 12-lead ECG, there are a few signs, symptoms, and presentations that strongly indicate non-cardiac etiologies. The first and easiest to identify is the patient’s age. It is highly unlikely that a patient who is under 30 years old who does not have a history of significant drug use, morbid obesity, diabetes, or other risk factors will have acute coronary syndrome. Also, pleuritic chest pain, that is chest pain that increases with coughing and inspiration, generally stems from pulmonary issues. Keep in mind that an elderly COPD patient may have chronic pleurisy and still suffer from ACS. Finally, pain that is reproducible by movement is more often musculoskeletal. As was previously mentioned, if you decided not to perform an ECG and it turns out that the patient was having an MI, you will be in a world of trouble so use your best judgment, don’t get lazy, and err on the side of caution.

The patients that require 12-lead ECGs have been identified, so now it’s time to discuss transmission. If you suspect STEMI or AMI and you have the ability to transmit the ECG to the receiving facility, you should do so. Studies show that transmission of the 12-lead ECG to the receiving facility has a positive impact on patient outcomes due to an increased likelihood of early activation on the cath lab and decreased door to balloon times. If you don’t have the ability to transmit the ECG the literature still shows that early ED notification reduces the in hospital time to fibrinolysis or percutaneous intervention. Be aware though that the same studies indicate that the physicians at the hospital are statistically more likely to start the process of activating the cath lab before you arrive at the hospital if the ECG has been transmitted to them. Some protocols like the Pennsylvania Department of Health ALS Suspected Acute Coronary Syndrome protocol recommend that all 12-lead ECGs recorded in the field should be transmitted to the receiving facility. The theory is that, if you were concerned enough to perform the ECG, then the patient may benefit from a physician reviewing it prior to your arrival at the emergency department.

**Proper 12-lead ECG Placement**

When performing a patient assessment, examination of the front of the patient’s body only would cause a lot of key information to be missed. That is because the body is three-dimensional. The heart is the same way, and the 12-lead ECG was developed to obtain a multidimensional view of its electrical activity. Each lead of the ECG traces the electrical conduction through a specific area of the
Standard Limb Lead Placement

Therefore, proper and consistent placement of the electrodes is imperative for accurate interpretation. The 12 leads of the ECG are divided into two groups. The first is the limb leads which provide a view of the frontal plane of the heart. The second is the precordial leads which show a view of the horizontal plane.

As you prepare the patient for the 12-lead ECG there are a number of things to keep in mind. The patient’s position will affect the tracing. If possible, the patient should be in the supine position but if he or she experiences dyspnea while flat, the head can be elevated to 30 degrees. Any patient movement while recording the ECG will cause artifact and make accurate interpretation more difficult. Therefore, if possible, make sure that the patient is warm and comfortable to prevent shivering or moving. Take note that oftentimes, the patient who is experiencing an acute myocardial infarction will be extremely anxious and it may be a formidable task for you to calm that patient enough to be still. Also, if you want an artifact free tracing, it would be in your best interest to perform the 12-lead ECG on scene or while the ambulance is parked. The electrodes must stick to the patient’s skin so you must be ready to prepare the area by drying it if the patient is diaphoretic or shaving the skin if the patient has significant body hair. Finally, once the leads are placed check your work and make sure they are in the correct location. It is very easy even for the most seasoned paramedic to make a mistake and place a lead in the incorrect location. Incorrect lead placement can cause ECGs that show false changes that can be misinterpreted as acute emergencies. There is nothing more embarrassing than diagnosing a STEMI and activating resources at the hospital only to find out that the reading was bad due to improper lead placement.

The limb leads show the frontal or vertical view of the heart. They measure electrical current as it moves from the shoulders to the feet. The limb leads are placed on the extremities or shoulders and hips. The vertical plane can be visually imagined if you place a circle on your patient that has a diameter stretching from the patient’s neck to the navel and from shoulder to shoulder. These leads are defined by their angle of orientation from the negative electrode to the positive electrode within the imaginary circle.

There are six limb leads and they are viewed by the variable designation of electrodes as positive and negative which is done by the ECG machine itself. Leads I, II, and III are the standard limb leads and form what is known as Einthoven's Triangle. Lead I has an angle of orientation of 0 degrees and the right arm electrode is positive and the left arm electrode is negative. If you picture the heart and place a straight line across the middle, that is lead I. Lead II has a direct view of the bottom of the heart with the right arm electrode negative and the leg electrodes positive. Lead II’s angle of orientation is +60 degrees. Lead III is a measure of the current as it passes from the left shoulder to the leg lead. It has an angle of orientation of +120 degrees.

Standard Limb Leads and the Einthoven Triangle

Leads AVL, AVR, and AVF are known as the augmented limb leads. They are drawn using the same electrodes as the standard limb leads. However, instead of having single negative and single positive leads as the standard ones, the augmented leads have a single positive lead and the rest are negative. The machine then draws an isoelectric line based on the average location of the negative leads. The “augmented” designation is given to these leads because the machine must amplify the tracing in order to have complexes that match the size of the other leads. That amplification is necessary because the machine is measuring an average current over a larger area as compared to the standard leads which measure a direct current. Lead AVL is a measure of the average electrical current moving to a positive electrode on the left arm. It has an angle of orientation of -30 degrees. Lead AVR is drawn when the right arm is made positive and has an angle of orientation of -150 degrees. Finally, lead AVF is drawn when the leg lead is...
positive and the average current moving from the head to the feet is measured. Lead AVF has an angle of orientation of +90 degrees.

**Precordial Lead Placement**

The six precordial leads or chest leads measure the electrical current in the horizontal or transverse plane and are arranged horizontally across the chest. Each individual electrode, V1-V6, is made positive in turn, with all the other leads being negative. These leads measure electric current moving anteriorly and posteriorly throughout the heart. V1 is placed in the fourth intercostal space to the right of the sternum. V2 is the mirror image of V1 and is placed in the fourth intercostal space to the left of the sternum. V4 is placed in the fifth intercostal space at the midclavicular line. V3 divides the space between V2 and V4. V6 is placed in the fifth intercostal space at the midaxillary line. Finally, V5 bisects the area between V4 and V6.

Now that the leads are placed on the patient the ECG is recorded. This is only half the battle. Once the ECG is obtained it is imperative to understand the anatomical location that each lead represents. Note that to be clinically significant of myocardial infarction changes in the ECG must be present in three or more consecutive leads. Lead II, III, and AVF show current passing through the inferior wall of the left ventricle. V1, V2, V3, V4, V5, and V6 show current moving through the septum, anterior wall, and lateral wall of the left ventricle. It is not uncommon to see infarctions that involve multiple areas of the precordial leads. For example, common site of infarction includes the anteroseptal wall, leads V1-V4, or anterolateral wall, and leads V3-V6. This occurs because of the common blood supply to these areas. Leads I and AVL also show the lateral wall of the left ventricle. AVR is not generally included in standard ECG interpretation. When you examine a standard ECG it is important to work in a systematic manner. Start at Lead II and move through III and AVF. Then look at V1-V6. The acronym I See All Leads can be a useful tool to remember the anatomic locations indicated by the leads:

- **I** - Inferior leads II, III and AVF
- **See** - Septal leads V1-V3
- **All** - Anterior leads V3-V4
- **Leads** - Lateral leads V5, V6, I, and AVL

You may have noticed that the standard lead placement only examines the left ventricle. That is because the vast majority of myocardial infarctions affect this area. However it is possible for a patient to experience an infarction on the right side of the heart. In fact, approximately 40% of patients who experience an inferior wall MI will also have right ventricular involvement. If you suspect a right sided infarction move V4 to the fifth intercostal space on the right side of the patient’s chest. This makes V4R. Some algorithms recommend performing V4R on all patients with inferior wall MIs. This will provide a view of the anterior wall of the right ventricle. You should be concerned of an infarction involving the right side of the heart if the patient presents with chest pain and hypotension. Remember, the cardiac anatomy that was discussed earlier. If the right ventricle is failing then there will be a decreased amount of blood available from the lungs for the left ventricle to pump into the systemic circulation. Also, jugular vein distension may be present due to a backflow of blood from the weakened right ventricle.

Before we move on to discussion of the 12-leads with acute changes and STEMI’s there are a few important notes on normal ECGs that must be considered. Interpretation of 12-lead ECGs is a skill that requires constant practice and you must be familiar with normal read outs. The 12-lead examines multiple views of the heart from different angles and plains, and therefore, the morphology of the normal complexes can change from lead to lead. In the left lateral and the inferior leads the P wave is normally small and positive. Note that a standard cardiac monitor commonly shows lead II, an inferior lead, with an angle of orientation of +60 degrees so the P wave is upright lasting .06 to .11 seconds on the strip. That same P wave when viewed from leads III and V1 is often biphasic because the angle of orientation is so different. In Lead AVR with its angle of orientation of -150 degrees the P wave is often a negative deflection completely. If you fail to remember the multi-view nature of the 12-lead you will surely find yourself confused. The QRS complex has similar properties. R waves with the largest positive deflection are found in the left lateral and inferior leads. Often there is a progressive increase in R wave size across the pericardial leads from V1 to V6. Non-pathological Q waves which are indicative of septal depolarization are not always visible but when they are present they are seen as a small negative depolarization in the left lateral leads. A normal septal (non-pathologic) Q wave will have an amplitude not greater than .1 mV. Finally, the T wave is generally a small positive deflection, especially in leads with tall R waves. Conversely, in leads like AVR where the R wave tends to be negative the T wave also tends to be negative. Individual patients will all have slightly different complex morphologies on their ECGs, and you must understand and be able to differentiate between normal variance and pathological change.
Identify Indicators of ACS on the 12-lead

When the blood supply to an area of the heart becomes occluded, ischemia, infarction, and necrosis occur and the 12-lead ECG is the most sensitive indicator of these changes even in the earliest phases of the cardiac event.\(^4\) A myocardial infarction develops over time and its progression is visible on the ECG.

The first signs of myocardial ischemia occur when a coronary vessel becomes occluded and visible changes to the T waves can be seen. The earliest sign is the peaking of the T waves where they become tall and narrow. These are also known as hyperacute T waves.\(^10\) Within a few hours these hyperacute T waves shrink and become inverted. Note that in these early stages of infarction, the patient may not be experiencing any pain or other symptoms. It is important to remember that T wave changes are a non-specific finding if they only occur in non-contiguous leads. Left ventricular hypertrophy may also cause T wave inversion; however, it is typically asymmetrical meaning that when you examine the T wave the first part of the negative deflection will be at a different angle than the return to the isoelectric line. When examining T waves, if they are inverted symmetrically and occur in contiguous leads, it is an ominous sign that should raise your index of suspicion for acute MI.

As the myocardial ischemia evolves into infarction, the ST segment will become elevated. Any patient that presents with a STEMI will need immediate intervention to prevent permanent damage to the myocardium. Be on the alert when treating a patient with significant ST elevation in multiple leads. His or her heart is failing and that patient may deteriorate into a ventricular dysrhythmia or cardiac arrest at any time. Finally, while any ST segment elevation should be concerning the 12 lead is only indicative of myocardial injury when the elevation is present in two or more contiguous leads.\(^2\)
If the patient survives the initial infarction without intervention then a pathological Q wave will be present on the ECG. The presence of a pathological Q wave is indicated by a large negative deflection prior to R wave. Specifically to be considered pathological the amplitude of the Q wave must be greater than or equal to one-third the amplitude of the following R wave in the same QRS complex. This forms a biphasic QRS complex in most leads. The one exception to this rule is in lead AVR where there is almost always a large Q wave. AVR should generally be ignored when assessing the ACS patient’s ECG. When a pathologic Q wave is present cellular necrosis is present and irreversible damage has occurred. Take note that a Q wave is only present when a transmural infarction occurs which involves the full thickness of the ventricular wall. If a subendocardial infarction occurs, that only involves a partial thickness of the ventricular wall. There will be the normal T wave changes and the ST elevation but no Q wave will be formed.

The heart is a three-dimensional structure and all of the anatomical aspects that the 12-lead ECG examines are connected. Hence it should be intuitive that when one area is damaged by an MI the other areas will be affected. These changes are known as reciprocal changes and they occur because of the net increase in electrical current in the areas that were unaffected by the infarction as the heart attempts to compensate. For example, when a patient experiences an inferior wall STEMI it is common to see contiguous symmetrical T wave inversions in the septal and anterior leads. If the infarction is large enough there can be reciprocal ST depression as well.

Bundle branch blocks occur when there is a disturbance in the interventricular electrical conduction system. They can present acutely or be present as a chronic condition. They are characterized by a widened and deformed QRS complex due to the delay in electrical conduction through the ventricles. These blocks are divided into two categories: blocks of the left bundle branch and blocks of the right bundle branch. Left bundle branch blocks (LBBB) are characterized by a QRS complex that is widened to greater than .12 seconds and a broad or notched R wave. There may be ST segment depression and T wave inversion. Right bundle branch blocks (RBBB) are also characterized by a QRS complex greater than .12 seconds. The R wave takes on a rabbit-eared formation that is also known as RSR prime and is present in leads V1 and V2. An RBBB may also have ST segment depression and T wave inversion. Often, if a patient is suffering from an acute left bundle branch block, it will be treated the same as a STEMI. However, if the patient has a chronic bundle branch block, it may be impossible to diagnose a STEMI in the field.
Another condition that can cause confusion on the 12-lead ECG is Wolff-Parkinson-White (WPW) Syndrome. This is a naturally occurring genetic disorder where there is a secondary pathway of electrical conduction from the atria to the ventricles. Most of the conduction will still occur through the AV node but some will pass through the aberrant pathway known as the Bundle of Kent. Often this condition is benign but it does cause changes on the ECG. The aberrant conduction from the atria to the ventricles in the WPW patient causes a widened QRS and a shortened PR interval. Oftentimes it will appear that the QRS complex arises right out of the P wave with a rapid upward swoop. This is designated as a Delta wave. In some leads this can be easily confused with a pathological Q wave. This chronically widened QRS also makes it near impossible to diagnose a bundle branch block from the ECG as well.

There are a number of conditions that can cause changes in the ECG that look similar to or can mimic MI but are not actually related to infarction. The disorders that will be discussed are Prinzmetal’s angina, hyperkalemia, hypothermia, the digitalis effect, and pericarditis. These are just a few examples and there are a multitude of other medical problems that can have similar effects. Keep in mind that any patient who has symptoms related to ACS and changes on the 12 lead ECG must be treated as if they are having an acute myocardial infarction until proven otherwise at the hospital.

The first mimicking condition is Prinzmetal’s angina which is caused by coronary artery spasm.10 The patient suffering from Prinzmetal’s angina will present in exactly the same way as the patient suffering from acute coronary syndrome. In fact, this patient will even show ST elevation on the 12-lead ECG. The only difference is that when given nitroglycerin the Prinzmetal’s angina patient’s ECG will return to normal. Nitroglycerin causes coronary artery dilation which immediately relieves the spasm and allows for immediate reperfusion of the affected area. Along with the normalization of the ECG the patient’s chest pain will likely be relieved completely which would be unusual if the patient was suffering from an infarction. Prinzmetal’s angina can occur in patients with underlying cardiovascular disease as well as patients who are otherwise healthy. There is no way to distinguish between these two groups in the field and it should not affect treatment.

Hyperkalemia, or a rise in serum potassium, is caused by a variety of conditions and it can cause significant changes on the ECG. As the potassium level rises the T waves rise in amplitude and become peaked. If the potassium level continues to rise, the PR interval elongates and the P wave becomes small and eventually will disappear. In the worst cases the QRS complex widens and merges with the T wave. If untreated ventricular fibrillation and cardiac arrest will eventually develop. In the early stages of hyperkalemia the peaked T waves can mimic ischemic T waves. The best way to distinguish between the two is that T wave changes related to ischemia will only be present in the leads covering the ischemic area where as hyperkalemic T waves will be diffuse and present in all leads.

Hypothermia that can cause ECG changes is defined as a core body temperature less than or equal to 30 degrees Celsius or 86 degrees Fahrenheit.2 When a patient is suffering from hypothermia everything slows down. You should expect to see bradycardia and prolonged intervals in all phases of the ECG. The change that can be confused with MI is the appearance of ST segment elevation that is known as an Osborn wave. The Osborn wave is characterized by a rapid and sharp ascent at the J point, the junction where the ST-segment takes off from the QRS complex, and then an equally sudden plunge back to the isoelectric line. As with the previously discussed electrolyte disturbances the ECG changes associated with hypothermia will diffuse throughout all leads of the ECG.

Digitalis is a naturally derived cardiac glycoside that is primary used in the treatment of atrial fibrillation and atrial flutter.11 The digitalis effect is a common normal, side effect of the drug on the patient’s ECG. It is characterized by ST segment depression and flattening or inversion of the T wave. The ST changes are generally most prominent in leads with a tall R wave.11

Finally, pericarditis, an infection of the pericardium, can cause ST elevation and T wave inversion that can be easily confused with evolving STEMI. It is also common for the patient to present with significant chest discomfort if the infection is acute. Treatment is generally the same, but keep in mind that nitroglycerin will have no effect on the pain and often the patient describes pain that is reproducible with movement.

**Management of the ACS Patient**

When treating the patient suffering from acute coronary syndrome the maxim is, “treat the patient not the monitor.” No matter what the ECG shows, if the patient is in distress, manage him or her in the same way that you would a patient who has a 12-lead with acute ST elevation.

If a patient presents with symptoms consistent with acute coronary syndrome obtain the 12-lead ECG before any other interventions. Doing this may increase sensitivity in detecting STEMI.4 There are also cases when the ECG will normalize after interventions such as the Prinzmetal’s angina that was discussed earlier. The earlier ECG is obtained the earlier it can be transmitted to the hospital so that the appropriate resources are mobilized. Remember that if the patient is also hypotensive, than a second ECG should be
obtained with V4r to assess for right sided infarction. If you transmit an ECG to the hospital that has a V4r reading on it make sure you notify medical command because the physician will not know that it is there just by looking at the strip. Throughout treatment and transport if you notice changes in the patient’s ECG it would be useful to obtain serial 12-leads to present to the receiving facility.

The patient should be placed on oxygen and the pulse oximetry should be monitored. If the patient’s SPO2 reading is normal on room air and he or she is not complaining of dyspnea then low flow O2 via nasal canula is acceptable. If the patient is experiencing respiratory distress or the SPO2 is less than 95% then he or she should be placed on high flow O2 via non-rebreather mask. Monitor lung sounds closely and be aware that a patient who has an infarction involving the left ventricle is at risk for acute pulmonary edema. Next, the patient should be given 324 mg of chewable aspirin. Aspirin is a platelet aggregation inhibitor and can prevent blood clots from forming and growing. There is a great deal of discussion concerning the administration of aspirin to chest pain/AMI patients who are compliant with a daily aspirin regimen. The Pennsylvania State ALS Protocol advises that if the patient has taken his or her daily dose of 324 mg of aspirin then no more should be administered. If the patient takes 81 mg of aspirin a day it is reasonable to give him or her another 243 mg orally. Make sure you follow your local protocols in regards to this medication administration.

Intravenous access is a priority and if the patient is hypotensive a fluid bolus should be administered. In order to perform catheterization or fibrinolysis in the hospital the staff will need two sites of vascular access. If secondary IV access can be obtained without delaying transport it can help to reduce door to reperfusion time upon arrival at the hospital. If your local protocols allow for adult intraosseous (IO) access it should be considered if intravenous access is not obtainable. Take into consideration the fact that if the patient is conscious, getting drilled with an IO needle will cause significant anxiety and fear which will further stress a cardiovascular system that is already struggling. Nitroglycerin causes coronary artery vasodilation. A dose of .4 mg should be administered sublingually if the patient is complaining of chest pain unless contraindicated. There are two primary contraindications for nitroglycerin and they are hypotension and the use of medications for erectile dysfunction. If a patient is already hypotensive it means that the cardiac output is already low and if nitrates are administered and the circulatory system becomes dilated it may cause total cardiovascular collapse. Viagra (sildenafil) and other erectile dysfunction (ED) medications increase blood flow to the penis by increasing systemic vasodilation. If they are administered in conjunction with nitrates a synergistic affect may occur. Take note that sildenafil, branded as Revatio, is prescribed to both men and women for pulmonary arterial hypertension. In combination, the two drugs sildenafil and nitroglycerin can have a significantly more potent effect than they do individually, increasing vasodilatation and causing cardiovascular collapse. Discussing the use of ED medication can be difficult for the patient and the provider but it is of the utmost importance. Some protocols allow for additional doses of nitroglycerin after the first one, administered q 3-5min as long as the patient’s systolic blood pressure remains greater than 100 mmHg. If the patient becomes hypotensive then a normal saline bolus should be administered until the patient’s systolic blood pressure returns to at least 100 mmHg. Make sure that you record vital signs after every dose of vasoactive medication.

After three doses of nitroglycerin if the patient is still experiencing chest pain a narcotic analgesic may be administered. The protocol that Pittsburgh Emergency Medical Services utilizes calls for morphine sulfate at a dose of 2-5 mg IV/IO that may be repeated every five minutes until the patient’s pain is controlled, a maximum dose of .2mg/kg is reached, or the patient becomes hypotensive. PA ALS Protocol also allows for 1 mcg/kg of fentanyl up to .2 mg/kg. For cardiac pain morphine may be a better choice because along with analgesia it has a greater vasodilatory and euphoric affect compared to fentanyl. Again, make sure you follow your local protocols with regard to the administration of narcotics. The most important thing that emergency medical services can do for the acute myocardial infarction is provide timely transport to the hospital. The best possible facility for treatment of the AMI patient is one capable of emergent catheterization. Studies show that bypassing community hospitals that are only capable of fibrinolytic therapy and administration of clot busting medication in order to triage patients to facilities capable of emergent percutaneous cardiac intervention is beneficial if the 911 to balloon time is less than 90-120 minutes. If you work in an urban EMS system this is easy, however, less than 25% of hospitals in the United States have the capability to perform emergent percutaneous intervention. Make your transport decision with your patient as well as your protocols in mind and remember that if you can get them to a designated heart attack center in a timely fashion they are more likely to have a positive outcome.

**Conclusion**

The ability of EMS professionals to identify and treat acute myocardial infarctions has grown significantly; however, there is still a great deal of room for improvement. For a paramedic to be able to accurately interpret a 12-lead ECG, constant practice is required. It is clear that early activation of in hospital resources has a positive impact on patient outcomes. Multiple studies have shown that without transmission of the ECG, physicians are hesitant to mobilize these resources based solely upon paramedic interpretation. There is also data that indicates that properly trained paramedics can accurately identify STEMI without physician assistance. This tells us two things. The first is that as paramedics we must be advocates for our patients and push for protocols that include automatic resource activation at the hospital. Second, if you have the ability to transmit the 12-lead, then do so. Finally, in the future look for prehospital fibrinolysis to come to a protocol near you. Studies in Europe have been ongoing for the last decade and have conclusively demonstrated that if prehospital professionals are equipped with clot busting drugs, time to treatment can be reduced significantly and thereby reduce morbidity and mortality. EMS professionals bring the medicine to the patient and we still make house calls!

**Terminology**

- **Angle of Orientation:** Is the orientation of an ECG lead from the negative electrode to the positive expressed in degrees by superimposing it on in on the 360 degree circle of the frontal plane. Lead I that travels from the right shoulder straight across to the left is designated as zero degrees.
- **Angina Pectoris:** The sudden onset of chest pain caused by myocardial ischemia. It is often described as retrosternal heaviness radiating to left arm and jaw.
- **Atherosclerosis** - A disorder characterized by the buildup of plaque, a combination of cholesterol and calcium in the lumen of the blood vessels. If untreated, it can lead to the occlusion of the vessel or the formation of a thrombus.
- **Aorta** - Emerging from the left ventricle it is the largest blood vessel in the body and it supplies oxygenated blood to the systemic circulation.
- **Atrioventricular (AV) Node** - The capacitor-like site in the right atrium adjacent to the septum that functions to slow and concentrate electrical impulses as they pass into the ventricles.
- **Automaticity** - The unique property of cardiac cells in which they are able to generate electrical current to stimulate muscular contraction without external stimulation.
- **AV Valves** - The valves that separate the atria and the ventricles. On the right side there is the tricuspid valve and on the left side there is the bicuspid mitral valve.
- **Biphasic Waves** - Waves on an electrocardiogram that contain both positive and negative deflections.
- **Bundle Branches** - Part of the electrical system of the heart that spreads electrical stimulation throughout the ventricles.
- **Bundle of His** - A continuation of the AV node.
- **Cardiac Output** - The amount of blood circulated throughout the body in one minute. It is generally expressed in liters per minute.
- **Circumflex Coronary Artery** - One of the main branches of the left coronary artery. It supplies blood to the left ventricle and intraventricular septum.
- **Coronary Arteries** - The arteries that supply the heart with oxygenated blood. A myocardial infarction occurs when one or more of these vessels becomes occluded.
- **Coronary Artery Disease** - A general description for the pathological process caused by atherosclerosis that leads to the narrowing and eventual occlusion of the coronary arteries.
- **Diastole** - The period of ventricular relaxation where the ventricles fill with blood that drains through the AV valves from the atria.
- **Depolarization** - The process where electrolytes move down the electrochemical gradient across the cell membrane creating an electrical impulse which ultimately stimulates muscular contraction.
- **Einthoven's Triangle** - The triangle formed around the heart by ECG leads I, II, and III.
- **Frank-Starling Mechanism** - Also described as the rubber band mechanism. It is the property of the heart muscle that when it is stretched the muscle contracts with greater force to a limit.
- **Infarction** - The necrosis or damage of an area of tissue caused by the occlusion of its blood supply.
- **Interventricular Septum** - The membrane that separates the left and right atrium.
- **Intraventricular Septum** - The membrane that separates the ventricles.
- **Ischemia** - When a particular area of tissue is oxygen starved. Ischemia precedes infarction and does not necessarily correlate to permanent damage.
- **Isoelectric Line** - The baseline of the electrocardiogram that is neither positive nor negative.
- **J Point** - The demarcation point between the QRS complex and the ST segment.
- **Left Anterior Descending Artery** - The other main branch of the left coronary artery. Also supplying blood to the left ventricle, the intraventricular septum, and sometimes the AV node.
- **Levine's Sign** - When a chest pain patient describes his or her pain by placing a closed fist on his or her chest. It is a strong indicator of myocardial ischemia.
- **Mediastinum** - The area in the chest between the lungs where the heart, aorta, and other major structures lie.
- **Osborn Wave** - A distinct type of the ST elevation seen in hypothermia that is characterized by a sharp ascent at the J point followed by an equally sharp return to the isoelectric line.
- **Preload** - The pressure in the cardiovascular system that is exerted upon the ventricles in their resting state when they are passively filling.
- **Purkinje Fibers** - A part of the electrical system of the heart that functions to conduct impulses from the bundle branches into the myocardium of the ventricles.
- **Reciprocal Changes** - Changes that occur on the STEMI patient's ECG in areas that are not infarcted.
- **Refractory Period (absolute and relative)** - The absolute refractory period occurs within a cell immediately after depolarization and in that period the cell cannot be stimulated at all because there is no electrochemical gradient present. The relative refractory period occurs next and in that period the cell can be stimulated but will produce a weakened response because repolarization is not complete.
- **Repolarization** - The process by which cardiac cells return to their resting potential after depolarization.
- **Semilunar Valves** - The aortic and pulmonic valves that separate the ventricles from the aorta and the pulmonary arteries.
- **Sinus Node** - The primary pacemaker in the healthy heart. It generates 60-100 beats per minute at rest and is located at the junction of the superior vena cava and the right atrium. It is innervated by the vagus nerve, hence the slowing affect of vagal stimulation.
- **Stroke Volume** - The amount of the blood that is pushed out of the ventricles by a single cardiac contraction.
- **Subendocardial Infarction** - An MI that only involves a partial thickness of the ventricular wall.
- **Systole** - The period during the cardiac cycle where the ventricles contract.
- **Thrombus** - A blood clot that has broken free from the lumen and travels through the circulation until it becomes lodged in a vessel causing an infarction.
- **Transmural Infarction** - An MI that involves the entire thickness of the ventricular wall. When this occurs, a pathological Q wave will be present.

References

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