Prehospital 12-Lead ECG

What You Should Know

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What You Should Know

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This booklet is intended for paramedics and other health care personnel operating in the prehospital environment who may be obtaining 12-lead ECGs on patients who are suspected of having an AMI. It is assumed the reader has basic skills in rhythm interpretation, a working knowledge of normal cardiac anatomy and physiology, and is familiar with the signs, symptoms and pathophysiology of acute myocardial infarction.

The objectives of this booklet include an introduction to the equipment and skills you will need to perform a 12-lead ECG (Chapter 1) and basic electrocardiography as it relates to waveform generation in the 12 ECG leads (Chapter 2). Electrocardiographic changes produced by myocardial infarction are discussed in Chapter 3. The final chapter provides sample 12-lead ECGs done by paramedics on actual patients. Use them to practice your newly acquired skills. Information provided in this booklet is for reference only and does not supersede institutional protocols, standing orders or device operating instructions. Always follow protocols and procedures authorized by your medical director. Readers interested in more advanced information on 12-lead ECG interpretation may refer to the texts

listed in the reference section of this booklet.

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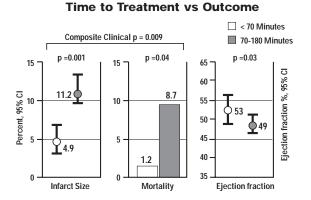


The 12-lead electrocardiogram (ECG) has traditionally been used in the hospital setting to help physicians identify, diagnose and treat patients with cardiac disorders. Advances in technology now make acquiring and transmitting 12-lead ECGs in the prehospital setting both feasible and beneficial. Prehospital 12-lead ECG with computer analysis and transmission to the emergency department is recommended by the American Heart Association (AHA) and the National Heart Attack Alert Program (NHAAP) for patients with chest pain and possible acute myocardial infarction (AMI) (1).

Nature of the Problem

Heart disease is a global medical problem. Although worldwide figures are not available, cardiovascular disease is the leading cause of death in the United States. It is estimated that each year 1.5 million Americans experience acute myocardial infarction and approximately one half million die. Half of these deaths occur outside of the hospital within the first hour of symptoms (2).

In addition, an AMI often has a negative impact on quality of life. These consequences can be reduced by the rapid identification and treatment of AMI. Rapid identification and treatment has been shown to preserve cardiac function, limit infarct size and reduce morbidity and mortality (3,4,5). In short, minimal time to treatment yields maximum patient benefit.



AMI patients treated within 70 minutes of chest pain onset experience significantly smaller infarcts, reduced mortality and higher ejection fractions (4).

Patients experiencing AMI symptoms often delay seeking medical care for a variety of cultural and personal reasons. This delay in conjunction with delays observed in the prehospital and hospital settings hinders prompt treatment of patients (6,7,8). The current clinical goal is to provide reperfusion therapy, if indicated, within 60 minutes of symptom onset (9) or within 30 to 60 minutes of patient arrival in the emergency department (1).

Communities, emergency medical services and hospital emergency departments are taking up the challenge to develop and implement strategies for reducing the time to treatment of patients with AMI.

Prehospital 12-Lead ECG—Part of the Solution

A 12-lead ECG performed and transmitted from the field is useful in the early detection and prompt treatment of patients with acute myocardial infarction. The procedure takes five minutes or less to perform and, when transmitted from the scene or en route, has been shown to shorten time to in-hospital treatment by roughly 30 to 60 minutes (10,11,12,13,14). The receiving ED can prepare for immediate patient assessment and rapid treatment with thrombolytic therapy or percutaneous transluminal coronary angioplasty (PTCA). Patients may also benefit from triage and transport to the most appropriate facility. Documentation of transient or intermittent arrhythmias and other electrophysiologic events that occur in the prehospital setting can assist in diagnosis and treatment decisions in the ED (14).

The prehospital 12-lead ECG offers paramedics and emergency physicians significant advantages over the single lead, cardiac monitoring typically available in EMS. The prehospital 12-lead ECG not only provides a diagnostic quality ECG¹ for use in the detection of AMI but also allows the knowledgeable paramedic to determine the area of myocardial injury, anticipate associated potential complications and implement treatment strategies accordingly (15,16). Additionally, it provides a baseline for serial ECG evaluations.

Equipment and Skills

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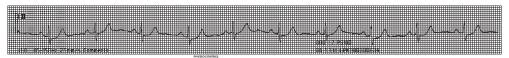
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12-Lead vs Single Lead Electrocardiography

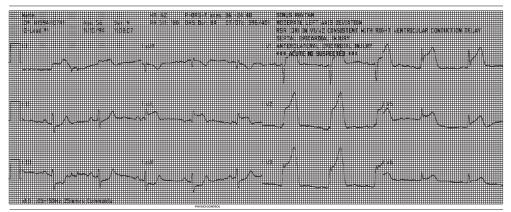
The electrocardiogram (ECG) is a noninvasive method of recording the electrical activity of the heart. When monitored in a single limb lead, for instance Lead II, only one view of these electrical events is represented and the information from other vantage points is hidden from the viewer.



An important clinical benefit of the 12-lead ECG stems from its ability to observe the heart's electrical activity from more than one angle. This can be readily appreciated if we liken the 12-lead ECG to having twelve different cameras positioned to record images of the heart from twelve different angles. Each angle, or lead, sees a partial view but each lead also misses a lot. The 12-lead ECG provides a more comprehensive way to evaluate the electrical activity of the heart.



Rhythm strip in lead II without evidence of AMI



12-lead ECG shows marked ST segment elevation indicative of AMI

As shown in the preceding ECGs, Lead II can look rather normal while marked ECG changes may be present in other leads. Here we see a rhythm strip of Lead II that gives no evidence of the dramatic ECG changes observable in Leads V1 through V5.

One may ask, if twelve leads are better than three, are more than twelve leads even better? While it is true that added views of the heart result in more information, practicality dictates that we limit the number of leads used in routine evaluations to a manageable number without compromising our ability to make an accurate diagnosis. The diagnostic value of 15 and 18 lead ECG is currently being evaluated in clinical research (17,18). These additional leads are positioned in order to view the right ventricle and the posterior wall of the left ventricle—areas of the heart that are subject to infarct but are not readily viewable by the standard 12-lead ECG.

Although 12-lead ECG is not the ultimate in electrocardiography, it has stood the test of time proving to be efficient and accurate when properly performed.

Equipment Requirements

Advances in technology have resulted in ECG monitors that can acquire 12-lead ECGs with ease. However, any ECG monitor that features a diagnostic frequency response for recording can be used to obtain diagnostic ECG tracings in multiple leads. A method of obtaining diagnostic 12-lead ECGs with a three lead device is described in Appendix A.

FREQUENCY RESPONSE

12-lead ECG recordings should always be performed in diagnostic frequency response. Diagnostic frequency response records ECG signals in a wide range (.05 to 150Hz) resulting in signal characteristics that would be filtered out in the narrower bandwidth of the common monitor frequency response (typically 1 to 30Hz or 0.5 to 40Hz).

To understand concept of frequencies and their relation to the ECG, consider music. High pitched, or treble sounds are actually high frequency sound waves. Low pitched, or bass sounds are sound waves that occur at low frequencies. When you adjust the treble or bass control on the stereo receiver you cause it to filter out certain frequencies along the spectrum of sound waves to suit your personal taste. The result is that some sound frequencies are authentically reproduced by the speakers, while others are damped or removed.

When the heart generates and conducts an impulse it produces an electrical signal much like the music in that it contains a broad spectrum of frequencies. The bandwidth or frequency response of an ECG monitor, like the stereo receiver, defines the range of frequencies that can be authentically reproduced on the display or the ECG recording. But unlike the stereo receiver the monitor offers a limited selection of bandwidths. In electrocardiography, the broad bandwidth is referred to as "diagnostic frequency response" or DIAG; the range of frequencies reliably reproduced is 0.05 (low end) to 100 or 150Hz (high end). Diagnostic frequency response is particularly important when trying to diagnose myocardial ischemia or injury by observing for abnormalities in the ST segment and/or T wave; these are low frequency signals.

- Use diagnostic frequency response (DIAG) for 12-lead ECGs
- DIAG bandwidth is 0.05 to 150Hz
- DIAG authentically reproduces ST segment on the ECG

A more narrow bandwidth called monitor frequency response (MON) will accurately reproduce frequencies between 0.5 to 40Hz or 1.0 to 30Hz depending on the device and the selections available. Monitor frequency response is very useful for routine ECG recording (rhythm strips) because it filters out much of the "noise" from muscle artifact (high frequency signals). The more narrow bandwidth also stabilizes the baseline which would otherwise "wander" when the device is subjected to a motion environment (a source of low frequency signals).

- Use monitor frequency response (MON) for rate and rhythm determinations
- MON bandwidth is 0.5 to 40Hz or 1 to 30Hz
- MON distorts ST segment on the ECG

Many monitor recorders have both monitor and diagnostic frequency response options available to the operator who can select the frequency response in which to record the cardiac signal. The monitor screen of these devices, however, is limited to monitor frequency response because of limitations in display technology, and the desire for less visible noise appearing on the screen.

Monitor frequency response is not suitable for determinations about ST segment elevation or depression because it does not reproduce the ST segment accurately on the recording or on the screen. In some cases it may show ST elevation or depression where none exists and in others diminish the degree or entirely cancel out the baseline deviation. Study the two recordings in figures A and B. They were taken on the same patient at nearly the same time. ST elevation is evident in the 3-lead rhythm strip recorded in monitor frequency response (fig. A); none appears in the 12-lead done in diagnostic frequency response (fig. B).

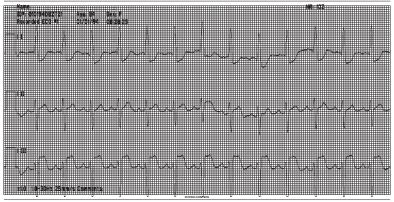


Figure A. Monitor frequency response. ST elevation in II/III; ST depression in I.

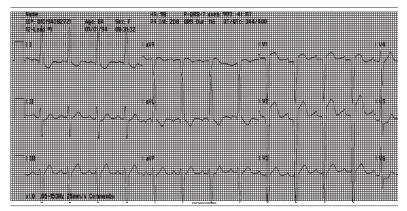


Figure B. Diagnostic frequency response. No ST elevation in II/III. ST depression moderated in I.

Figure C is an ECG recording from another patient. The ECG trace to the left of the dashed line was recorded in monitor frequency response. The operator switched to a DIAG recording at the point of the dotted line. ST segment elevation is evident only in the DIAG portion of the recording.

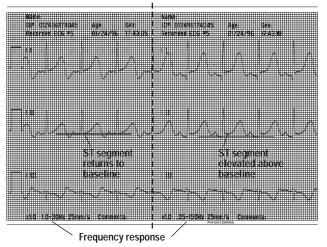


Figure C. Monitor frequency response does not accurately reproduce the ST segment.

This phenomenon is explained by the amplitude (height) and direction of the QRS deflection, positive or negative, in the lead you are observing. In monitor frequency response, the beginning of the ST segment will be distorted in the direction opposite of the predominant QRS deflection.

In figure A, the QRS complex in lead I is mostly positive and the ST segment is distorted downward. The QRS complex in lead III is mostly negative and the ST segment is distorted upward. In figure B, the diagnostic recording gives an authentic representation of the ST segment; ST segment distortion is not present in either lead I or III.

The same phenomenon can be seen in figure C but it is more subtle. The DIAG ECG recording (to the right of the dotted line) shows ST segment elevation of about 2mm in lead II. The QRS complex is positive in lead II. When the ECG is viewed in monitor frequency response (to the left of the dotted line), distortion causes the ST segment to appear at the baseline.

In summary, since monitor frequency response does not reproduce the ST segment accurately, it should not be used to determine the presence or absence of ST segment changes indicative of myocardial ischemia or acute myocardial infarction. Monitor frequency response is useful in the reduction

of spurious artifact from motion and vibration and is adequate when viewing the ECG for cardiac rate and rhythm. For more authentic ECG recordings or diagnostic 12-lead ECGs, be sure to use the DIAG feature of the ECG device.

TELECOMMUNICATIONS

Cellular telephone communications has made it possible to transmit 12-lead ECGs from the scene or en route to a receiving device stationed at an emergency department or other patient care facility. Standard telephone lines may be used where cellular technology is not available or when the 12-lead is acquired in a residence or workplace where there is easy access to an standard phone line.

Use of a cellular telephone is made convenient and easy due to its similarities with standard landline telephones. Although a cellular phone acts like a standard telephone, it is in fact a two-way radio. Two-way radios emit signals that can disrupt the function of susceptible medical devices (electromagnetic interference or "EMI"). Cellular technology and its use in the hospital setting is a controversial issue. Published information on cellular phone usage and its potential interference with medical devices is growing, as is recognition that these concerns must be addressed². Tests have shown that interference varies from device to device and area to area. Generally, devices placed in close proximity — up to three feet — to a radio are more likely to be affected although some devices may continue to be vulnerable at distances of 12 to 15 feet. It is advisable to develop a policy on the use of cellular phones particularly in the vicinity of life support equipment.

Features and system requirements for transmission of ECG data differ depending on the device used. Refer to the device operating instructions for details on transmission features and requirements.

Selecting Patients for Prehospital 12-Lead ECG

In 1992 the American Heart Association published updated recommendations for early treatment of patients with chest pain and possible AMI. Prehospital screening for thrombolytic therapy and prehospital 12-lead ECG were included as optional guidelines in the revised AMI algorithm for Advanced Cardiac Life Support providers (1). Sample checklists and protocols, which incorporate these guidelines, may be found in Appendix B. Obtaining a 12-lead ECG when an AMI is suspected is obviously warranted and beneficial. There are also other situations in which a 12-lead may be useful. Some patients have infrequent, transient, and/or atypical cardiac symptoms or events which make accurate diagnosis difficult. For example, the recording of ischemic ECG changes before treating angina with nitroglycerin may be the only opportunity to document on ECG that your patient is experiencing chest pain of cardiac origin. Likewise, 12-lead ECG documentation of arrhythmias can be helpful in the diagnosis and treatment of patients who exhibit transient syncope or palpitations.

Patients who exhibit symptoms of congestive heart failure, pulmonary edema, sudden onset of respiratory distress or unexplained diaphoresis are also good candidates for a 12-lead ECG as it may provide clues to the patient's condition.

Asymptomatic ischemia or infarct can be detected if a 12-lead ECG is performed on patients at risk for this condition. Consider performing a 12-lead ECG on patients with a history of coronary artery disease (CAD) or risks for CAD (diabetics, the elderly, smokers), especially when their presenting problem results in a decrease in myocardial blood supply or an increase in myocardial oxygen demand. For example, any patient with CAD who is tachycardic, hypoxemic or who has significant blood loss from any cause would be a candidate for 12-lead ECG. Of course, the decision to perform 12-lead ECG by EMS providers is governed by local protocol.

Preparing the Patient

Both preparation of the skin to remove oils and dead skin cells and the elimination of muscle tension are important in obtaining a noise free recording. Cleanse the skin with an alcohol pad followed by a brisk rub with a piece of dry rough material like a towel or a piece of gauze. Position the patient's arms and legs in a comfortable position in which the extremities are resting on a supportive surface. Any self-support of limbs by the patient may introduce fine muscle artifact even though the patient does not appear to be moving. Attaching the electrode to the leadwire prior to placing the electrodes on the patient is more comfortable for the patient and preserves the integrity of the electrode gel.

Electrode Placement for 12-Lead ECG

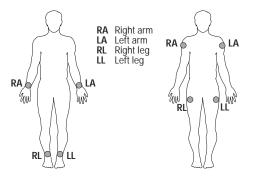
Six of the 12 ECG leads, Leads I, II, III, aVR, aVL and aVF, are derived from signals obtained from four electrodes placed on the limbs. The

remaining six leads, V1, V2, V3, V4, V5 and V6, are derived from six electrodes placed on the left side of the chest. Accordingly, the standard 12-lead ECG cable contains ten leadwires. These leadwires are typically labeled: LA, RA, RL, LL, V1, V2, V3, V4, V5 and V6. Each labeled leadwire should match the electrode site identified on the patient.

Correct electrode placement is important for accurate interpretation of the 12-lead ECG and for meaningful comparisons between the prehospital 12-lead and any other 12-leads acquired previously on the patient. Begin by removing or loosening patient clothing from around the wrists and ankles and from the left side of the chest. The chest must be sufficiently exposed to determine anatomical landmarks for the precordial electrodes.

LIMB LEAD ELECTRODES

Limb lead electrodes are typically placed on the inside of the wrists and ankles, however, these electrodes may be placed anywhere along the limbs. Limb lead electrodes should NOT be placed on the torso when performing a 12-lead ECG. Placement of the RA, LA, RL and LL electrodes on the torso for a 12-lead ECG will result in a non-standard 12-lead. The impact of non-standard limb lead placement on the accuracy of computerized interpretive statements such as ***ACUTE MI SUSPECTED*** has not been determined.



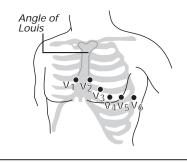
Although the electrodes may be placed anywhere along the limbs, standard locations for limb lead electrodes are:

- Right arm electrode near the wrist on the inside of the patient's right arm;
- Left arm electrode near the left wrist;
- Left leg electrode on the inner aspect of the left leg near the ankle;
- Right leg electrode near the right ankle.

Alternate placement on the upper arms and thighs may be helpful in reducing artifact.

PRECORDIAL LEAD ELECTRODES

Electrode placement for precordial leads (leads V1 through V6) will be described in detail. For the best learning experience, practice finding the landmarks on yourself and others with differing body types.



Precordial lead electrode placement Lead Location

- V1 Fourth intercostal space to the right of the sternum.
- V2 Fourth intercostal space to the left of the sternum.
- V3 Directly between leads V2 and V4.
- V4 Fifth intercostal space at midclavicular line.
- V5 Level with V4 at left anterior axillary line.
- V6 Level with V5 at left midaxillary line (directly under the midpoint of the armpit).

The electrode placement for lead V1 is in the fourth intercostal space to the right of the sternum. To locate the V1 position, place your index finger in the notch at the top of the sternum. Move your finger down the sternum until you feel an elevation or thickening of the bone. This is the angle of Louis (pronounced Louie) where the manubrium joins the body of the sternum. Move your finger down and to the patient's right into the depression between the second and third ribs. This is the second intercostal space. Continue to palpate down the right side of the sternum two more spaces. You have located the fourth intercostal space. Apply the electrode and leadwire for V1.

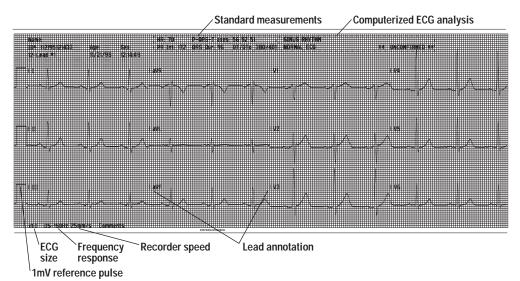
Move across the sternum to the fourth intercostal space on the patient's left side. Apply the electrode and leadwire for V2. V4 is located next because it provides a reference point for V3. V4 is located in the fifth intercostal space, at the midclavicular line. Find the fifth intercostal space by palpating for the next intercostal space below the V2 position. Then follow this space with your index finger laterally to intersect with the midclavicular line. Place the V4 electrode at this point. Place V3 halfway between leads V2 and V4. Place the V5 electrode level with V4 at the left anterior axillary line. Place the V6 electrode level with V5 at the left midaxillary line.

When performing 12-lead ECGs on female patients, place the electrodes for leads V3 through V6 under rather than on the breast.

Once all the connections are made, encourage the patient to lie quietly for the data collection period. Resting the patient's arms and legs on a supportive surface will minimize the amount of noise introduced into the ECG. For details on acquiring a 12-lead ECG with your device, refer to the device operating instructions.

Layout of the 12-Lead ECG

Commonly, the 12-lead ECG report is printed in the format shown below. The six limb leads (I, II, III, aVR, aVL and aVF) are ordered in two groups of three on the left side of the report while the six precordial leads (V1, V2, V3, V4, V5 and V6) are ordered in two groups of three on the right side of the report.



Some devices print standard measurements such as the QRS axis and ECG intervals at the top of the report. The intervals are often reported in milliseconds. In this example, the PR interval is reported as 172 (ms). For instructions on converting milliseconds to seconds, see Chapter 2.

The report may also include a 1mV reference pulse, an annotation of the ECG size setting, the frequency response and the recorder speed. The 12-lead ECG is usually printed in x 1.0 size in which 1mV=10mm (two large boxes) on the ECG paper. The standard diagnostic frequency response is .05 to 150Hz. Recorder speed is typically 25mm/sec. Consistency in the areas of frequency response, ECG size and recorder speed is important when interpreting a 12-lead ECG and comparing reports.

Computerized ECG Analysis

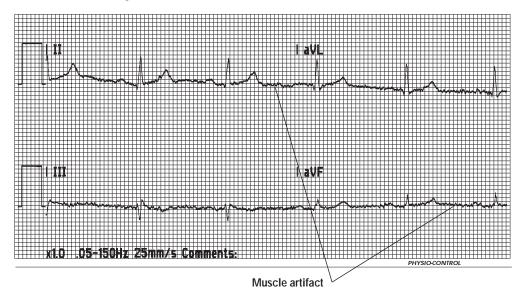
Some 12-lead ECG devices are capable of providing an interpretation of the patient's cardiac rhythm and analyzing normal or abnormal ECG findings. To do this, the device records the patient's 12-lead ECG and examines it against an internal set of criteria and rules of logic. The result is a computerized ECG analysis or "interpretive" 12-lead ECG. The 12-lead ECG should be "overread" by a physician prior to making a decision to withhold or prescribe patient treatment. Sensitivity and specificity for AMI detection will differ between computerized systems. Refer to the manufacturer's product specifications and description of the computerized ECG algorithm for details.

Common Technical Problems

Three common types of artifact are muscle artifact, wandering baseline and electromagnetic interference (EMI). The presence of artifact may interfere with the device's ability to acquire and/or interpret the 12-lead ECG.

MUSCLE ARTIFACT

Muscle artifact is a high frequency, low amplitude, somewhat irregular distortion of the baseline and QRS complexes. It is often due to poorly supported or tense muscles in the arms and legs. Muscle artifact may be mimicked by loose, dry or outdated electrodes, a loose lead wire or poor patient cable connection, poor skin preparation and vibration from a moving vehicle.

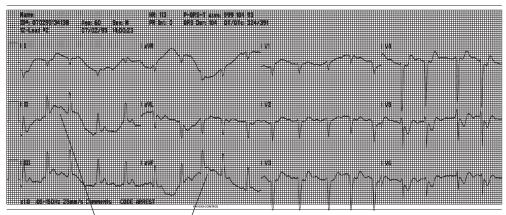


Possible solutions:

- Keep electrodes in unopened package until time of use
- Use electrodes before expiration date
- Perform skin prep
- Rest arms and legs on a supportive surface
- Ensure patient comfort, coach patient to relax
- Ensure good patient/electrode contact, replace dry electrodes
- Secure cable connections
- Stop vehicle while acquiring 12-lead ECG

WANDERING BASELINE

Wandering baseline is a low frequency, high amplitude artifact. It may be observed during deep inhalations and exhalations or during the acceleration and deceleration forces of a moving vehicle.



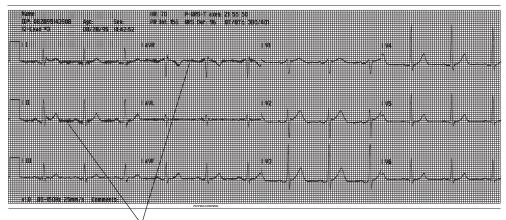
Wandering baseline

Possible solutions:

- Ask patient to breathe quietly while acquiring 12-lead ECG
- Eliminate all patient movement
- Perform good skin preparation
- Check all electrodes for good contact
- Stop vehicle while acquiring 12-lead ECG

ELECTROMAGNETIC INTERFERENCE (EMI)

Electromagnetic interference originates outside the patient and is caused by interference from electronic sources such as power cords, AC powered equipment that may be insufficiently grounded, and near-by use of a hand held radio. This is also called AC (alternating current) or 60 cycle³ interference.



Electromagnetic interference (EMI)

Possible solutions:

- Move away from noisy environment
- Ensure power cords are not touching or lying near patient cable
- Move away from or unplug AC powered equipment that is causing interference
- If using the Auxilliary Power Supply module, switch the monitor to battery power
- Check lead wires and patient cable for observable damage

ECG Measurements and Waveforms

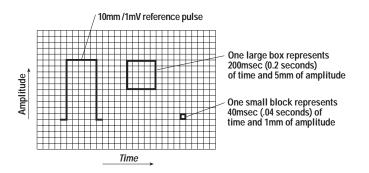
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Assessment of a 12-lead ECG requires familiarity with the ECG graph paper, normal intervals and waveform morphology. This chapter reviews some basic principles of recording electrical signals generated by the heart.

ECG Paper

The horizontal axis of the ECG grid is used to measure time (duration) and the vertical axis is used to measure amplitude (voltage). One horizontal small block is 1mm in width and equals 40 milliseconds of time (.04 seconds) when the standard 25mm/sec recorder speed is used.

One vertical small block is 1mm in amplitude and equals 0.1mV when the standard ECG size (x1.0) is used. ECG size x1.0 is calibrated to 10mm/ 1mV as depicted by the 1mV reference pulse. These amplitude rules apply to both conventional three lead ECG monitoring and 12-lead ECGs.

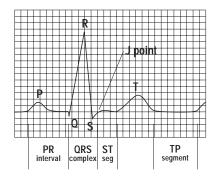


Time intervals and durations, such as the PR interval and QRS duration, are generally printed in milliseconds on the 12-lead ECG. If you have memorized the normal intervals and durations in fractions of seconds, you will need to adjust your thinking about these measurements. To convert milliseconds to seconds, move the decimal point three places to the left.

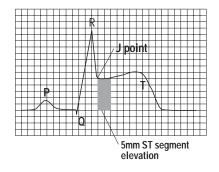
$$200ms = .200. = .20$$
 seconds
 $80ms = .080. = .08$ seconds

Intervals and Segments

The PR interval and QRS duration are used to assess for conduction delays while the TP segment and the J point are important in the evaluation of ST segment elevation. The TP segment extends from the end of the T wave of one beat to the beginning of the P wave in the next beat. It is a good place to establish the baseline. The J point marks the transition from the QRS complex to the ST segment.

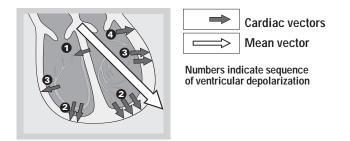


ST segment elevation is the hallmark of myocardial injury on the 12-lead ECG. ST segments are considered elevated if the J point falls \geq 1mm above the baseline.



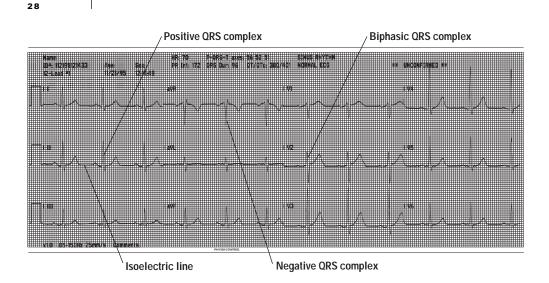
Generating an ECG Waveform

As electrical depolarization spreads throughout the ventricular myocardium, many small electrical currents are formed which follow different paths. These paths are referred to as vectors. The initial vector (1) moves from left to right as the intraventricular septum depolarizes. The right and left ventricles depolarize next (2, 3 and 4). The net electrical activity on the left is greater than that which occurs on the right because of the larger amount of left ventricular muscle mass. One general direction of depolarization predominates when the individual vectors are combined. This is termed the mean vector. During depolarization in the normal heart, the mean vector moves downward and leftward.

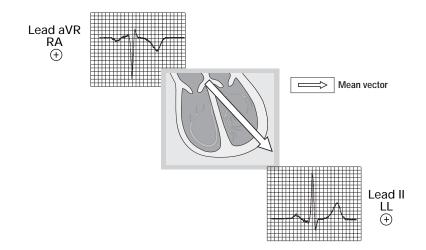


Electrodes attached to the external surface of the body record the net direction of travel and the net amplitude of vectors generated during depolarization. Each of the 12-leads has one positive electrode which "sees" depolarization from its unique perspective. Depolarization traveling toward a positive electrode results in a positive deflection. When depolarization travels away from a positive electrode, a negative deflection results. If depolarization travels perpendicular to the electrode an equiphasic (or biphasic) waveform results. Electrically silent periods produce an isoelectric (flat) line. This is referred to as the baseline.





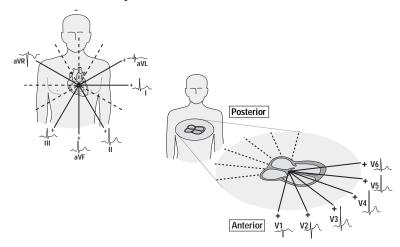
Observe the QRS complexes in Leads II and aVR. The QRS complex in Lead II is positive (upright) because the mean vector of depolarization travels toward the left leg (LL) which is the positive electrode in Lead II. The QRS complex in Lead aVR is negative because the mean vector travels away from the right arm (RA) which is the positive electrode in Lead aVR.



The 12 ECG Leads

The 12 ECG leads are made up of six limb leads (lead I, II, III, aVR, aVL and aVF) which record the ECG in the frontal plane of the heart and six chest or precordial leads (leads V1, V2, V3, V4, V5 and V6) which record the ECG in the horizontal plane of the heart.

The frontal plane provides information about current flowing both up and down and right to left within the heart. The horizontal plane provides information on the frontward (anterior) and backward (posterior) current flow. Since each of the 12 ECG leads views the heart's electrical activity from a different angle, each lead writes its own distinctive pattern of cardiac electrical activity on the ECG.



LIMB LEADS

The limb leads are derived from electrodes placed on the extremities right arm (RA), left arm (LA), and left leg (LL)—and recording the voltages between them. A fourth electrode applied to the right leg (RL) serves to reduce noise common to all the electrodes—a technique known as "common mode rejection." Three of the limb leads are bipolar leads and the remaining three are unipolar leads.

The bipolar limb leads

Leads I, II, and III are the bipolar leads. A bipolar lead has one negative and one positive electrode. The ECG machine automatically assigns the proper polarity (negative or positive) to the electrodes for each recorded lead. The ECG in a bipolar lead is a recording of the voltage differences between the two active electrodes during the cardiac cycle.

	RA	LA	LL	RL
Lead I	-	+		
Lead II	-		+	
Lead III		-	+	
inactive electrode common mode rejection				

The unipolar limb leads

Leads aVR, aVL and aVF are the unipolar limb leads. The letter "a" stands for augmented, the "V" stands for voltage and the letters "R," "L" and "F" refer to the location of the positive electrode. In aVR the positive electrode is RA, in aVL the positive electrode is LA, and in aVF the positive electrode is LL (F is substituted for "foot").

A unipolar lead has a positive electrode but no negative electrode. Instead, the voltages detected at the two remaining electrode sites are used by the device to automatically derive an electrical reference point called the central terminal (CT). The ECG in a unipolar lead is a recording of the

	RA	LA	LL	RL
Lead aVR	+			
Lead aVL		+		
Lead aVF			+	
СТ	common mode rejection			

30

voltage differences between the CT and the positive electrode during the cardiac cycle.

PRECORDIAL LEADS

The six precordial leads, labeled V1, V2, V3, V4, V5 and V6, are unipolar much like the unipolar limb leads. Each precordial electrode acts as the positive electrode for its lead. For example, the V1 electrode is the positive electrode for lead V1. The ECG machine automatically assigns the positive polarity to the appropriate electrode for each precordial lead and calculates a different reference point known as Wilson's terminal. Theoretically, Wilson's terminal represents an electrode positioned at the very center of the heart.

Together, the 12 ECG leads provide information about the inferior (II, III, aVF), lateral (I, aVL, V5, V6) septal (V1, V2) and anterior (V3, V4) surfaces of the left ventricle. Lead aVR rarely provides information relevant to infarction. ECG signals from the right ventricle and the posterior wall of the left ventricle are not well represented in the standard 12-lead ECG.

Identifying ECG Changes

- 35 Ischemia, Injury and Infarct Defined
- 36 ECG Changes with Ischemia, Injury and Infarct
- 39 Using ECG Leads to Localize Ischemia, Injury and Infarct
- Coronary Artery Distribution and Associated Clinical Findings
- 46 Right Sided Chest Leads
- 47 ST Elevation in Conditions Other than AMI
- 48 Conclusion

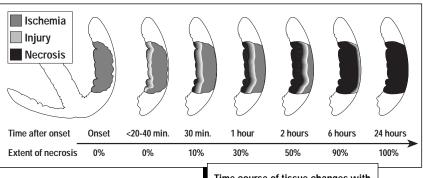
The 12-lead ECG is an important tool in the diagnosis of myocardial ischemia, injury and infarction. By the end of this chapter, you should be able to localize the infarct and determine what coronary artery is affected.

Ischemia, Injury and Infarct Defined

Before describing the ECG findings for these conditions, it is necessary to define the terms. In ischemia, blood flow to an area of the heart is insufficient to meet the oxygen requirements of the heart muscle. This generally occurs as a result of narrowed coronary arteries. If blood flow is increased or oxygen requirements are lowered, ischemia will resolve without permanent damage.

Ischemia is also present in the initial minutes after a total coronary artery occlusion. When a coronary artery remains occluded by a thrombus or from prolonged coronary artery spasm, myocardial ischemia progresses to injury. This mechanism of injury represents the hyperacute phase of myocardial infarction. Injured heart muscle cannot properly conduct electrical impulses nor contract properly. Injured myocardium can be rescued if the blockage can be alleviated quickly and blood flow restored. If blood flow is not restored, the heart develops an area of infarct. Infarcted myocardium is dead. It does not conduct electrical impulses and it does not contract. The damage of infarct is irreversible.

Although the onset of symptoms may be sudden and acute, myocardial infarction is not a sudden and complete event. It evolves over time from the first seconds of ischemia to the early minutes of injury (20 to 40 minutes) to the later stages of infarct (two to twelve hours). After six hours, myocardial infarction is usually 90% complete.



Time course of tissue changes with ischemia, injury and infarct.

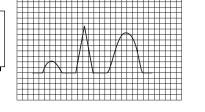
ECG Changes with Ischemia, Injury and Infarct

Ischemia, injury and infarct are usually, but not always, evident on the 12-lead ECG. As cardiac ischemia evolves to injury and infarct, the ECG changes also evolve.⁴ For this reason, patients admitted for suspected AMI undergo a series of 12-lead ECGs (in addition to other tests) over a period of hours to days.

ISCHEMIA

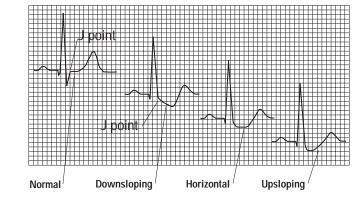
Ischemia presents itself as changes to the ST segment and the T wave. The ischemic T wave is often peaked and symmetrical and may be either upright or deeply inverted.

Schematic of ischemic T waves

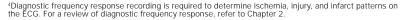


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ST segment depression however, is the most common and specific hallmark of ischemia. ST segments are considered depressed when they measure \geq 1mm (one small box) below the baseline at the J point. The J point, the juncture of the QRS complex and the ST segment, may not always be clearly visible. ST segment depression may be downsloping, horizontal or upsloping—the horizontal and downsloping types are usually indicative of myocardial ischemia.



Normal and ischemic ST segments

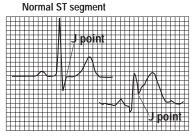


ST segment depression may also be associated with left ventricular hypertrophy, left bundle branch block and digitalis in therapeutic and toxic doses. Therefore, the patient's clinical presentation must be considered when reading the 12-lead ECG since ST segment depression may be associated with one of the conditions noted above rather than due to ischemia.

INJURY

Injury usually presents on the 12-lead ECG as ST segment elevation within 20 to 40 minutes of coronary artery occlusion. ST segment elevation is a "red flag" of AMI. It is particularly pertinent to the prehospital setting since it often develops during the time when patients experiencing AMI symptoms will call for emergency care. AMI patients with ST segment elevation, when treated promptly with thrombolytic agents or PTCA, often have smaller infarcts and fewer complications (3,4,5).

ST segments are considered elevated when they measure \geq 1mm above the baseline at the J point. When measuring the amount of ST elevation, it is important to use the TP segment to establish the baseline. Using the PR segment as the baseline can lead to faulty measurements of ST elevation.

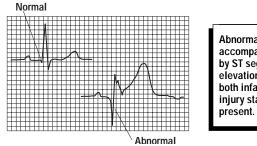


Abnormal ST segment



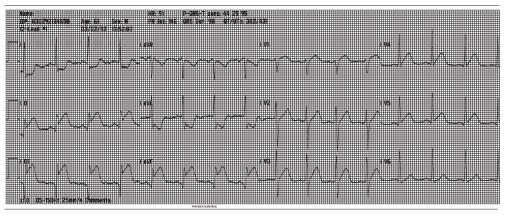
INFARCT

The presence of abnormal Q waves on the 12-lead ECG is an indication of infarcted (dead) myocardium. Abnormal Q waves generally are absent in the early evolution of AMI and appear only after several hours to days. Abnormal Q waves are \geq 40ms wide and/or are \geq 25% of the height of the R wave in that lead.



Abnormal Q wave accompanied by ST segment elevation indicates both infarct and injury stages are present.

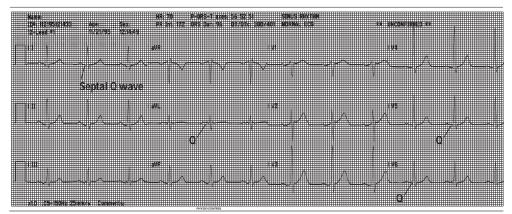
The presence of abnormal Q waves is solely a marker for the presence of dead myocardium. They do not, by themselves, indicate when the infarct occurred. Patients exhibiting abnormal Q waves may have suffered infarction several hours ago, several months ago or several years ago. Abnormal Q waves combined with inverted T waves or ST segment elevation, however, may indicate a recent (hours to days) infarction. Once they have developed, abnormal Q waves are unlikely to disappear.



12-lead with ST segment elevation and abnormal Q waves in leads II, III, and aVF

While abnormal Q waves indicate infarcted muscle, shallow, narrow Q waves are normal in some leads (I, aVL, V5 and V6). Normal Q waves represent depolarization of the intraventricular septum. A normal "septal"

Q wave is less than 40ms wide (one small box) and less than 25% of the height of the R wave in that lead.

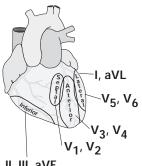


12-lead with normal "septal" Q waves in leads I, aVL, V5 and V6

Using ECG Leads to Localize Ischemia, **Injury and Infarct**

Localizing the area of injury on the 12-lead ECG is clinically useful in anticipating certain associated potential complications. It is important to remember, however, that the ECG can be normal in a patient experiencing myocardial infarction. The ECG changes described below are those that are most likely to occur during the course of injury and infarction.

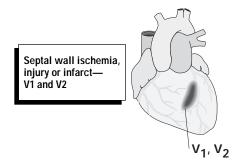
The left ventricle of the heart has thick muscular walls that are named according to how the heart lies in the chest and the leads of the 12-lead ECG are grouped according to the regions of the left ventricle they view. Leads V1 and V2 face the SEPTAL wall of the left ventricle, leads V3 and V4 face the ANTERIOR wall, leads II, III and aVF face the INFERIOR wall, and leads I, aVL, V5 and V6 face the LATERAL wall. None of the conventional 12 ECG leads directly view the right ventricle nor the POSTERIOR wall of the left ventricle.



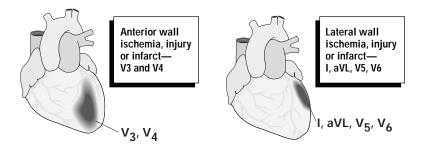
II, III, aVF

39

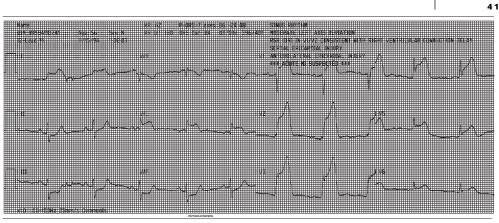
ST elevation of 1mm or more observed in two or more anatomically contiguous ECG leads is a "red flag" for the treatable injury phase of AMI. Anatomically contiguous leads are those leads that face the same area of the heart. For instance, myocardial injury sustained only in the SEPTAL area may be seen on the ECG as ST segment elevation in leads V1 and V2.



Localized ANTERIOR wall injury may elicit ST elevation in leads V3 and V4 while LATERAL wall injury generally appears in leads V5 and V6 and possibly leads I and aVL.

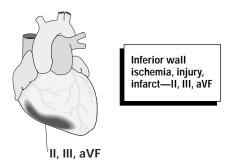


Of course, injury patterns may be more widespread in larger infarcts for instance ANTEROSEPTAL with ECG changes in V1 through V4 and ANTEROLATERAL with changes in V3 through V6. Consider the following ECG from a patient with extensive anterior wall injury. Note the relatively normal waveform in lead II.



12-lead ECG with > 1mm ST segment elevation in leads I, aVL, V1-V5

During INFERIOR wall injury, ST segment elevation is usually present in leads II, III, and aVF.



In summary, the 12-lead ECG provides views of the septal, anterior, lateral and inferior walls of the left ventricle. There are at least two leads that look at each of the areas. If ECG changes are present in one lead, confirm the changes are also present in other leads for that area. Remember that ST elevation of 1mm or more observed in two or more anatomically contiguous ECG leads is a "red flag" for the treatable injury phase of AMI.

I	lateral	aVR	V1 septal	V4 anterior
Ш	inferior	aVL lateral	V2 septal	V5 lateral
III	inferior	aVF inferior	V3 anterior	V6 lateral

Quick reference for localizing 12-lead ECG changes

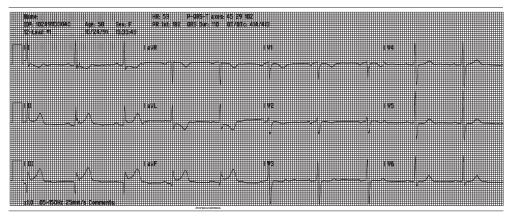
RECIPROCAL ECG LEADS

Reciprocal leads are those that do not view an area of the heart directly but may reflect a mirror image of the direct ECG changes.

For instance, none of the conventional 12 ECG leads directly face the POSTERIOR wall⁵, however, reciprocal ECG changes may be seen in the ANTERIOR leads when the POSTERIOR wall is affected. For instance, ST depression in the ANTERIOR leads usually mirrors the ST elevation that would otherwise be viewed in a POSTERIOR lead. A list of reciprocal lead groups is provided.

Site	Facing Leads	Reciprocal Leads
Septal Anterior Anteroseptal Lateral Anterolateral Inferior Wall Posterior Wall	V1-V2 V3-V4 V1-V4 I, aVL and V5 or V6 I, aVL and V3-V4 II, III and aVF None	None None II, III and aVF II, III and aVF I and aVL V1-V4

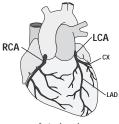
In the ECG below, ST elevation is evident in the inferior leads (II, III, aVF). Note the ST segment depression in the lateral leads (I, aVL). This could be a sign of lateral ischemia but is more likely a reciprocal change due to the inferior wall injury.



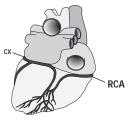
ST segment depression in leads I and aVL is reciprocal to the ST elevation in the inferior leads

Coronary Artery Distribution and Associated Clinical Findings

The heart muscle receives its blood supply from two major coronary arteries and their branches. Ischemia, injury and infarct develop within a region of the left ventricle (and to a lesser degree the right ventricle) according to the location of the coronary artery occlusion preventing distribution of oxygenated blood to heart muscle downstream. Coronary anatomy is not entirely consistent among all individuals but generally speaking the ANTERIOR, LATERAL, and SEPTAL walls of the left ventricle are supplied by the left coronary artery (LCA) and its branches while the right ventricle and the INFERIOR wall of the left ventricle are supplied by branches of the right coronary artery (RCA). Blood supply to the POSTERIOR wall of the left ventricle is shared by the distal branches of both the left and right coronary arteries.



Anterior view

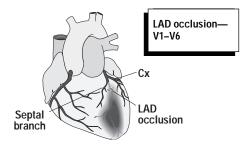


Posterior view

LEFT CORONARY ARTERY

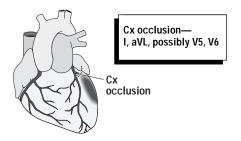
The left coronary artery branches into the left anterior descending (LAD) and the circumflex artery (Cx). The LAD travels over the ANTERIOR wall of the heart, supplying the anterior wall of the left ventricle, the interventricular septum, and the Bundle of His and bundle branches.

The LAD splits into the septal branch which supplies the intraventricular septum and the diagonal branches which supply the LATERAL wall of the left ventricle. Occlusions to the LAD or its branches result in ECG changes in the precordial leads (V1–V6).



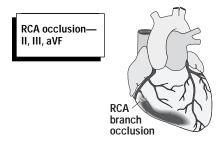
LAD occlusions may provoke the clinical complications of congestive heart failure, right and left bundle branch blocks, complete heart block and premature ventricular contractions. Heart block associated with LAD occlusions may not respond to atropine and are likely to require transthoracic pacing.

The Circumflex (Cx) artery travels between the left atrium and the left ventricle, supplying the left atrium, the POSTERIOR wall of the left ventricle and high LATERAL portions of the left ventricle. In some people, the Cx (rather than the RCA) supplies the AV node. LATERAL wall infarcts associated with Cx artery occlusion show ECG changes in leads I and aVL and possibly V5 and V6. Infarction is not usually isolated to the lateral wall but may be extensions of anterior, inferior or posterior MIs.

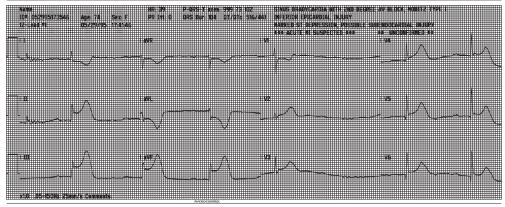


RIGHT CORONARY ARTERY

In 90% of the population the right coronary artery (RCA) travels between the right atrium and right ventricle branching to supply the INFERIOR wall of the left ventricle, the right ventricle, the right atrium, the sino-atrial (SA) and atrio-ventricular (AV) nodes, and the POSTERIOR wall of the left ventricle. Evidence of RCA occlusion and inferior wall MI on ECG will appear in Leads II, III and aVF, the INFERIOR leads.



Patients with RCA occlusion can develop sinus node disturbances such as sinus bradycardia and sinus arrest and AV blocks of varying degree. Noninvasive pacing or drug therapy may be required if the patient becomes symptomatic. Atrial arrhythmias such as premature atrial beats, atrial flutter and atrial fibrillation can also develop.



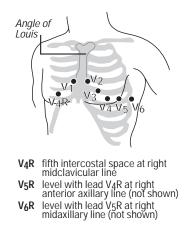
12-lead with bradycardia, AV block and ST elevation in inferior leads-suspect RCA occlusion

If you identify acute inferior wall injury or infarct, it is wise to also suspect a right ventricular infarct. Right ventricular infarction occurs in 30 to 40% of left ventricular inferior wall infarctions. To look for ECG changes indicative of right ventricular infarctions, you must observe one or more additional leads recorded from the right side of the chest.

Right Sided Chest Leads

There is increasing use of right sided chest leads to directly observe for ST segment elevation indicative of right ventricular infarction. Right ventricular infarction should always be suspected when ST segment elevation in leads II, III and aVF is present.

To obtain the right sided chest leads designated V4R, V5R and V6R, remove the V4, V5 and V6 leadwires from the left chest and attach fresh electrodes. Position the electrodes using the same anatomic landmarks on the right side of the chest. If you wish to use only one right sided lead, lead V4R is the most likely to detect right ventricular ECG changes.



The ECG machine cannot recognize that the electrodes have been moved to the right chest. Add an "R" to the lead annotations on the 12-lead strip to indicate the origin of the trace.

Unless right sided chest leads are available, 12-lead ECGs of patients with right ventricular infarction may often be indistinguishable from patients with uncomplicated inferior wall myocardial infarction. Yet these patients may require different management for hypotension and pain control. If the RV infarct patient exhibits hypotension, fluid therapy which increases preload and improves the pumping function of the right ventricle may be warranted. Therapies that reduce preload (morphine, nitroglycer-ine) should be used with caution when a right ventricular infarction is suspected. Some protocols allow for the special needs of the right ventricular infarct patient (see Appendix B, page 81).

ST Elevation in Conditions Other than AMI

There are some conditions in which ST segments appear elevated but AMI is not present. These include pericarditis, left bundle branch block, rhythms generated by an electronic ventricular pacemaker, left ventricular hypertrophy, and early repolarization. It is beyond the scope of this booklet to discuss each of these conditions in detail. However, some hints to their recognition are presented.

ST elevation in all leads, or nearly all leads supports the diagnosis of pericarditis. Pericarditis is an inflammation of the membranous sac that surrounds the heart. Pericarditis can be identified by the presence of a pericardial rub on auscultation. Pericardial pain is often relieved by having the patient lean forward while seated.

In left bundle branch block, the delayed and abnormal depolarization of the left ventricle with its wide and bizarre shaped QRS complex masks the ST segment elevation of AMI. Therefore, it is not possible to diagnose AMI using the 12-lead ECG in these patients. Left bundle branch block is identified by a wide QRS complex (≥ 120msec) and a predominately negative QRS deflection in lead V1.

A left bundle branch block pattern is typically seen in patients with an implanted pacemaker. If the heart is totally driven by the pacemaker, it is not possible to evaluate the ECG for evidence of ischemia, injury or infarct.

Left ventricular hypertrophy (LVH) is the enlargement of the left ventricle. Patients with LVH have increased QRS amplitude and ST and T wave abnormalities. The ST segments may be elevated or depressed and the T wave may be in the opposite direction of the QRS complex.

Early repolarization also presents as ST segment elevation on the 12-lead ECG. Early repolarization is a normal ECG variant which often occurs in young, healthy individuals, particularly black men. Early repolarization can frequently be differentiated from the injury pattern by the concave shape of the ST segment. This pattern is often seen in the lateral leads.

Despite the fact that AMI patients can have normal 12-leads and that ST segment elevation occurs in conditions other than AMI, don't despair. Accurate diagnosis of the patient with chest pain is a challenge for even the experienced physician. Diagnosis often requires more information than a 12-lead ECG alone. However, the astute prehospital provider can successfully add 12-lead to his or her overall patient assessment and report those abnormal findings that appear on the 12-lead. This strategy promotes the goal of early detection and rapid treatment of the patient with AMI.

Conclusion

This concludes the teaching content of this booklet. Patient selection for 12-lead ECG, performing proper electrode placement and acquiring a 12-lead ECG have been discussed. Topics of myocardial ischemia, injury and infarct and their associated ECG changes have been described. Some suggestions have been offered for anticipating patient complications depending on the location of the AMI.

The real learning begins when you perform your first 12-lead ECG and systematically evaluate the waveforms. A suggested format and sample ECGs in the next chapter provide an opportunity to practice interpretive skills.

A thorough analysis of the 12-lead ECG is more involved than this booklet describes. For more advanced material on 12-lead ECG interpretation, consult one of the texts listed in the reference section.

A Prehospital Approach to 12-Lead ECG Interpretation

- 52 Tutorial
- 54 Practice ECGs
- 72 Answer Key

This chapter describes a method for rapid interpretation of the 12-lead ECG adapted for the prehospital setting. Although the method emphasizes detection of ischemia, injury and infarct, it is important to include an assessment of cardiac rate and rhythm.

Rapid recognition of ischemia or evolving myocardial infarction is vital to early treatment and improved patient outcomes. Because of this, certain ECG patterns should be learned to the point where they become "red flags."

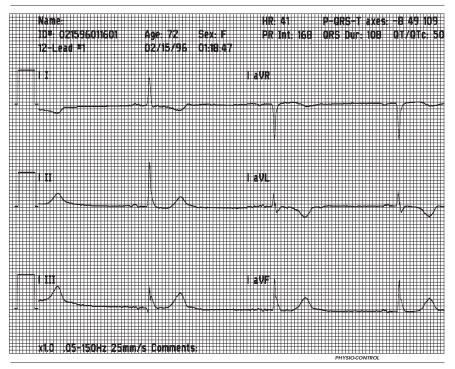
Reminder: These ECG changes occurring in two anatomically contiguous leads are significant:

- T wave inversion indicates ischemia; normal in lead aVR
- ST segment depression indicates ischemia
- ST segment elevation (ST [↑]) indicates acute injury
- Abnormal Q waves with ST↑ indicates acute infarct
- Abnormal Q waves without ST[↑] indicates a previous infarct, its age is not identifiable

"Reading" 12-lead ECGs requires the use of a consistent approach so the reader develops skill in pattern recognition. Frequent repetition of a standardized approach is the best learning tool. Use the steps provided to become familiar with prehospital 12-lead ECG interpretation.

It is important not to overly rely on the computerized ECG interpretation. The best use of this feature, if it is available, is to use it as a second opinion to your own impressions. Always confirm your findings of ECG abnormalities with a physician skilled in 12-lead ECG interpretation.





Step 1 Confirm proper technique

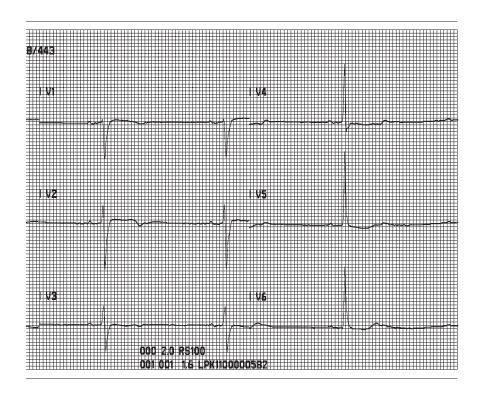
Frequency response set to DIAG (.05 to 100 or 150Hz)
 ECG size set to x1.0

Step 2a Scan each of the leads for T wave inversion (**ischemia**). On the chart below, mark an X in the leads where T wave inversion occurs.

Step 2b Scan each lead for ST segment depression (**ischemia**) and mark the chart.

Step 2c Scan for ST segment elevation (injury), mark the chart.

	I	Ш	Ш	aVR	aVL	aVF	V1	V2	V3	V4	V5	V6
T wave inversion (T↓)	X			to the second	X		X	X	X			
ST segment depression (ST↓)												
ST segment elevation (ST1)												
Abnormal Q waves (abn Q)												



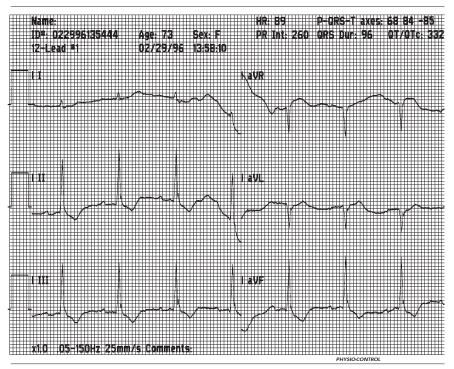
<u> $T\downarrow$ </u> Septal (V1, V2) <u> $T\downarrow$ </u> Lateral (I, aVL, V5, V6)

<u> $T\downarrow$ </u> Anterior (V3, V4) <u>Inferior</u> (II, III, aVF)

Step 4 Consider other conditions that can result in ST segment elevation. If no ST elevation is present, skip to Step 5.

<u>Skip</u> LBBB (Hint: QRS duration \geq 120ms and complex is
negative in Lead V1. Can't make AMI diagnosis by ECG.)
Electronic ventricular pacemaker rhythm
Pericarditis
Left ventricular hypertrophy
$__ \downarrow$ Early repolarization

normal ECG	acute infarct
septal and ischemia	previous infarct
anterolateral	condition that mimics
acute injury	acute MI on ECG





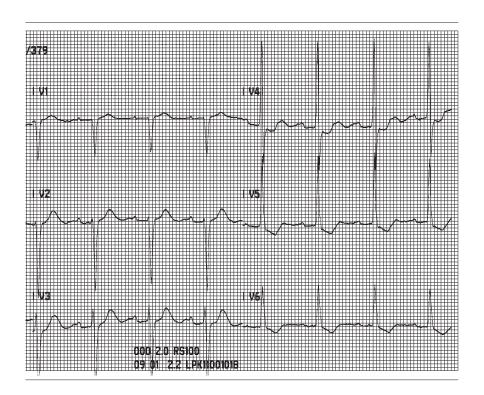
Frequency response set to DIAG (.05 to 100 or 150Hz)
 ECG size set to x1.0

Step 2a Scan each of the leads for T wave inversion (**ischemia**). On the chart below, mark an X in the leads where T wave inversion occurs.

Step 2b Scan each lead for ST segment depression (**ischemia**) and mark the chart.

Step 2c Scan for ST segment elevation (injury), mark the chart.

	Ι	Ш	III	aVR	aVL	aVF	V1	V2	V3	V4	V5	V6
T wave inversion (T↓)												
ST segment depression (ST↓)												
ST segment elevation (ST1)												
Abnormal Q waves (abn Q)												



_____ Septal (V1, V2) _____ Lateral (I, aVL, V5, V6)

_____ Anterior (V3, V4) ______ Inferior (II, III, aVF)

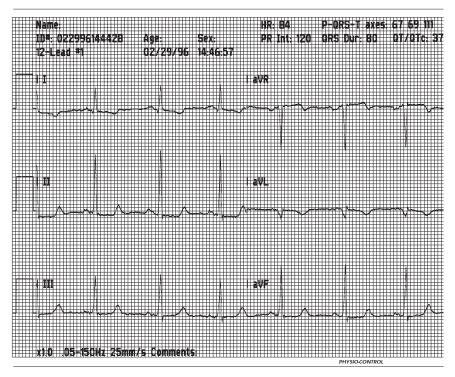
Step 4 Consider other conditions that can result in ST segment elevation. If no ST elevation is present, skip to Step 5.

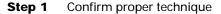
LBBB (Hint: QRS duration ≥ 120ms and complex is negative in Lead V1. Can't make AMI diagnosis by ECG.)
Electronic ventricular pacemaker rhythm
Pericarditis

_____ Left ventricular hypertrophy

_____ Early repolarization

normal ECG	previous infarct
ischemia	condition that mimics
acute injury	acute MI on ECG
acute infarct	





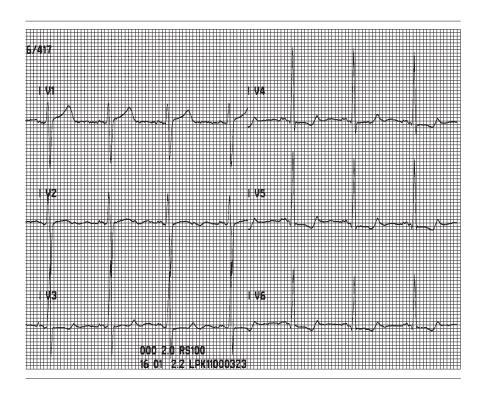
Frequency response set to DIAG (.05 to 100 or 150Hz)
 ECG size set to x1.0

Step 2a Scan each of the leads for T wave inversion (**ischemia**). On the chart below, mark an X in the leads where T wave inversion occurs.

Step 2b Scan each lead for ST segment depression (**ischemia**) and mark the chart.

Step 2c Scan for ST segment elevation (injury), mark the chart.

	Ι	Ш	111	aVR	aVL	aVF	V1	V2	V3	V4	V5	V6
T wave inversion (T↓)												
ST segment depression (ST↓)												
ST segment elevation (ST1)												
Abnormal Q waves (abn Q)												



_____ Septal (V1, V2) _____ Lateral (I, aVL, V5, V6)

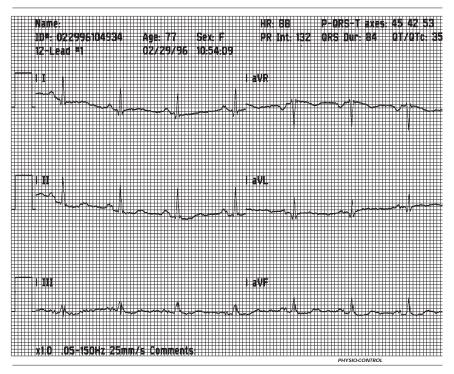
_____ Anterior (V3, V4) ______ Inferior (II, III, aVF)

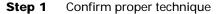
Step 4 Consider other conditions that can result in ST segment elevation. If no ST elevation is present, skip to Step 5.

- LBBB (Hint: QRS duration ≥ 120ms and complex is negative in Lead V1. Can't make AMI diagnosis by ECG.)

 Electronic ventricular pacemaker rhythm
- _____ Pericarditis
- _____ Left ventricular hypertrophy
- _____ Early repolarization

normal ECG	previous infarct
ischemia	condition that mimics
acute injury	acute MI on ECG
acute infarct	





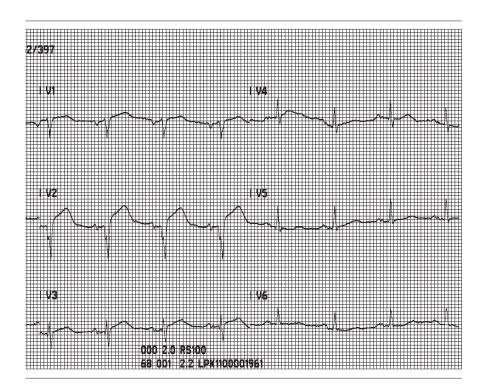
Frequency response set to DIAG (.05 to 100 or 150Hz)
 ECG size set to x1.0

Step 2a Scan each of the leads for T wave inversion (**ischemia**). On the chart below, mark an X in the leads where T wave inversion occurs.

Step 2b Scan each lead for ST segment depression (**ischemia**) and mark the chart.

Step 2c Scan for ST segment elevation (injury), mark the chart.

	Ι	Ш	111	aVR	aVL	aVF	V1	V2	V3	V4	V5	V6
T wave inversion (T↓)												
ST segment depression (ST↓)												
ST segment elevation (ST1)												
Abnormal Q waves (abn Q)												



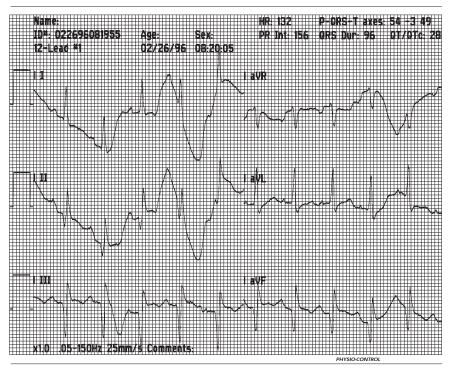
_____ Septal (V1, V2) _____ Lateral (I, aVL, V5, V6)

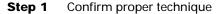
_____ Anterior (V3, V4) ______ Inferior (II, III, aVF)

Step 4 Consider other conditions that can result in ST segment elevation. If no ST elevation is present, skip to Step 5.

- LBBB (Hint: QRS duration ≥ 120ms and complex is negative in Lead V1. Can't make AMI diagnosis by ECG.)
 Electronic ventricular pacemaker rhythm
 Pericarditis
- _____ Pericarditis
- _____ Left ventricular hypertrophy
- _____ Early repolarization

normal ECG	previous infarct
ischemia	condition that mimics
acute injury	acute MI on ECG
acute infarct	





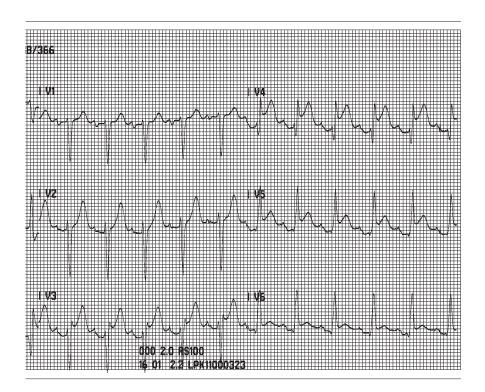
Frequency response set to DIAG (.05 to 100 or 150Hz)
 ECG size set to x1.0

Step 2a Scan each of the leads for T wave inversion (**ischemia**). On the chart below, mark an X in the leads where T wave inversion occurs.

Step 2b Scan each lead for ST segment depression (**ischemia**) and mark the chart.

Step 2c Scan for ST segment elevation (injury), mark the chart.

	Ι	Ш	111	aVR	aVL	aVF	V1	V2	V3	V4	V5	V6
T wave inversion (T↓)												
ST segment depression (ST↓)												
ST segment elevation (ST1)												
Abnormal Q waves (abn Q)												



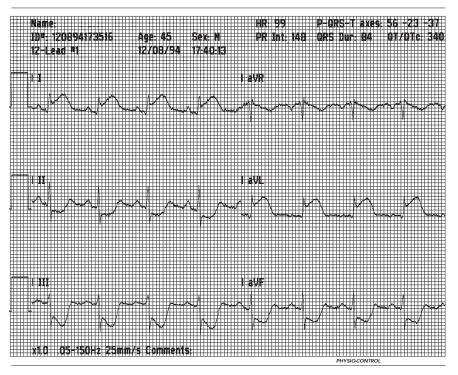
_____ Septal (V1, V2) _____ Lateral (I, aVL, V5, V6)

_____ Anterior (V3, V4) ______ Inferior (II, III, aVF)

Step 4 Consider other conditions that can result in ST segment elevation. If no ST elevation is present, skip to Step 5.

- LBBB (Hint: QRS duration ≥ 120ms and complex is negative in Lead V1. Can't make AMI diagnosis by ECG.)
 Electronic ventricular pacemaker rhythm
 Pericarditis
- _____ Left ventricular hypertrophy
- _____ Early repolarization

normal ECG	previous infarct
ischemia	condition that mimics
acute injury	acute MI on ECG
acute infarct	





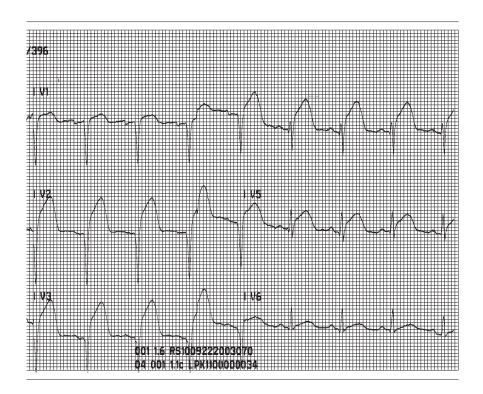
Frequency response set to DIAG (.05 to 100 or 150Hz)
 ECG size set to x1.0

Step 2a Scan each of the leads for T wave inversion (**ischemia**). On the chart below, mark an X in the leads where T wave inversion occurs.

Step 2b Scan each lead for ST segment depression (**ischemia**) and mark the chart.

Step 2c Scan for ST segment elevation (injury), mark the chart.

	Ι	Ш	111	aVR	aVL	aVF	V1	V2	V3	V4	V5	V6
T wave inversion (T↓)												
ST segment depression (ST↓)												
ST segment elevation (ST1)												
Abnormal Q waves (abn Q)												



_____ Septal (V1, V2) _____ Lateral (I, aVL, V5, V6)

_____ Anterior (V3, V4) _____ Inferior (II, III, aVF)

Step 4 Consider other conditions that can result in ST segment elevation. If no ST elevation is present, skip to Step 5.

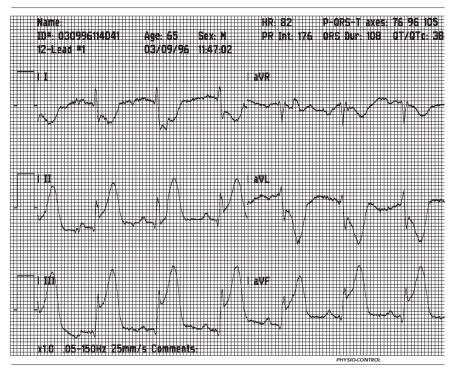
- LBBB (Hint: QRS duration ≥ 120ms and complex is negative in Lead V1. Can't make AMI diagnosis by ECG.)

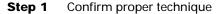
 Electronic ventricular pacemaker rhythm
- _____ Pericarditis

_____ Left ventricular hypertrophy

_____ Early repolarization

normal ECG	previous infarct
ischemia	condition that mimics
acute injury	acute MI on ECG
acute infarct	





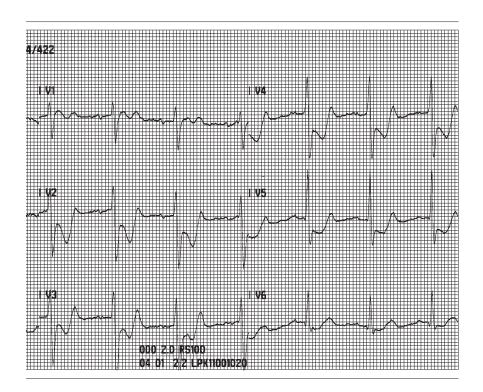
Frequency response set to DIAG (.05 to 100 or 150Hz)
 ECG size set to x1.0

Step 2a Scan each of the leads for T wave inversion (**ischemia**). On the chart below, mark an X in the leads where T wave inversion occurs.

Step 2b Scan each lead for ST segment depression (**ischemia**) and mark the chart.

Step 2c Scan for ST segment elevation (injury), mark the chart.

	Ι	Ш	111	aVR	aVL	aVF	V1	V2	V3	V4	V5	V6
T wave inversion (T↓)												
ST segment depression (ST↓)												
ST segment elevation (ST1)												
Abnormal Q waves (abn Q)												



_____ Septal (V1, V2) _____ Lateral (I, aVL, V5, V6)

_____ Anterior (V3, V4) ______ Inferior (II, III, aVF)

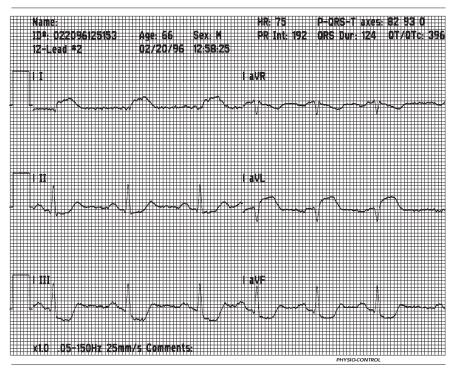
Step 4 Consider other conditions that can result in ST segment elevation. If no ST elevation is present, skip to Step 5.

LBBB (Hint: QRS duration ≥ 120ms and complex is negative in Lead V1. Can't make AMI diagnosis by ECG.)
Electronic ventricular pacemaker rhythm
Pericarditis

_____ Left ventricular hypertrophy

_____ Early repolarization

normal ECG	previous infarct
ischemia	condition that mimics
acute injury	acute MI on ECG
acute infarct	





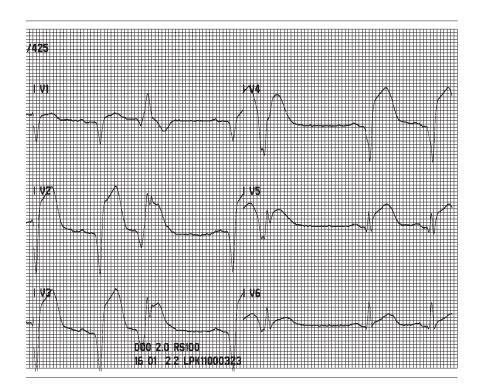
Frequency response set to DIAG (.05 to 100 or 150Hz)
 ECG size set to x1.0

Step 2a Scan each of the leads for T wave inversion (**ischemia**). On the chart below, mark an X in the leads where T wave inversion occurs.

Step 2b Scan each lead for ST segment depression (**ischemia**) and mark the chart.

Step 2c Scan for ST segment elevation (injury), mark the chart.

	Ι	Ш	111	aVR	aVL	aVF	V1	V2	V3	V4	V5	V6
T wave inversion (T↓)												
ST segment depression (ST↓)												
ST segment elevation (ST1)												
Abnormal Q waves (abn Q)												



Step 3 Localize the ECG changes to an area of the heart. Indicate type of change (i.e. $T\downarrow$, $ST\downarrow$, $ST\uparrow$, abn Q)

_____ Septal (V1, V2) _____ Lateral (I, aVL, V5, V6)

_____ Anterior (V3, V4) ______ Inferior (II, III, aVF)

Step 4 Consider other conditions that can result in ST segment elevation. If no ST elevation is present, skip to Step 5.

LBBB (Hint: QRS duration ≥ 120ms and complex is negative in Lead V1. Can't make AMI diagnosis by ECG.)
Electronic ventricular pacemaker rhythm
Pericarditis

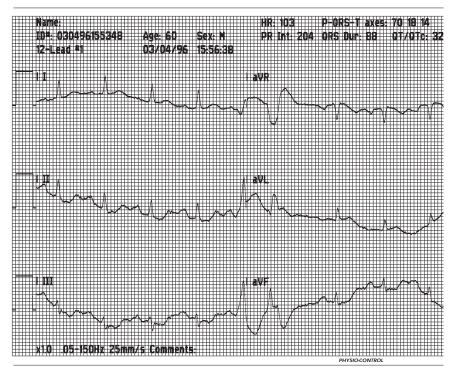
_____ Left ventricular hypertrophy

_____ Early repolarization

Step 5 State your impressions and request confirmation/consultation with a physician experienced in 12-lead ECG interpretation. Include the type of condition suspected and the area of the heart affected.

normal ECG	previous infarct
ischemia	condition that mimics
acute injury	acute MI on ECG
acute infarct	

Practice ECG #8





Frequency response set to DIAG (.05 to 100 or 150Hz)
 ECG size set to x1.0

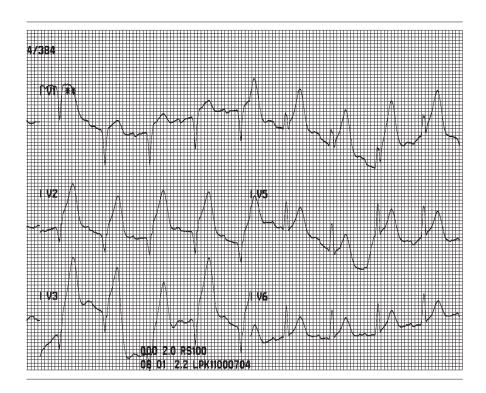
Step 2a Scan each of the leads for T wave inversion (**ischemia**). On the chart below, mark an X in the leads where T wave inversion occurs.

Step 2b Scan each lead for ST segment depression (**ischemia**) and mark the chart.

Step 2c Scan for ST segment elevation (injury), mark the chart.

Step 2d Scan for abnormal Q waves (infarct), mark the chart.

	Ι	Ш	111	aVR	aVL	aVF	V1	V2	V3	V4	V5	V6
T wave inversion (T↓)												
ST segment depression (ST↓)												
ST segment elevation (ST1)												
Abnormal Q waves (abn Q)												



Step 3 Localize the ECG changes to an area of the heart. Indicate type of change (i.e. $T\downarrow$, $ST\downarrow$, $ST\uparrow$, abn Q)

_____ Septal (V1, V2) _____ Lateral (I, aVL, V5, V6)

_____ Anterior (V3, V4) ______ Inferior (II, III, aVF)

Step 4 Consider other conditions that can result in ST segment elevation. If no ST elevation is present, skip to Step 5.

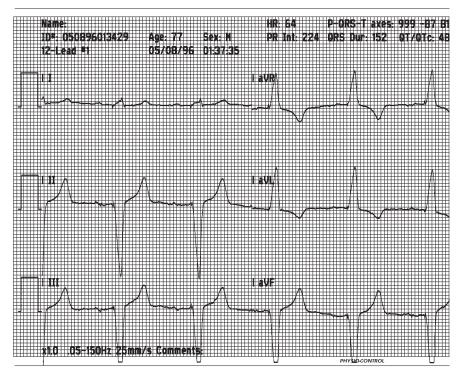
- LBBB (Hint: QRS duration ≥ 120ms and complex is negative in Lead V1. Can't make AMI diagnosis by ECG.)

 Electronic ventricular pacemaker rhythm
- _____ Pericarditis
- _____ Left ventricular hypertrophy
- _____ Early repolarization

Step 5 State your impressions and request confirmation/consultation with a physician experienced in 12-lead ECG interpretation. Include the type of condition suspected and the area of the heart affected.

normal ECG	previous infarct
ischemia	condition that mimics
acute injury	acute MI on ECG
acute infarct	

Practice ECG #9





Frequency response set to DIAG (.05 to 100 or 150Hz) ECG size set to x1.0

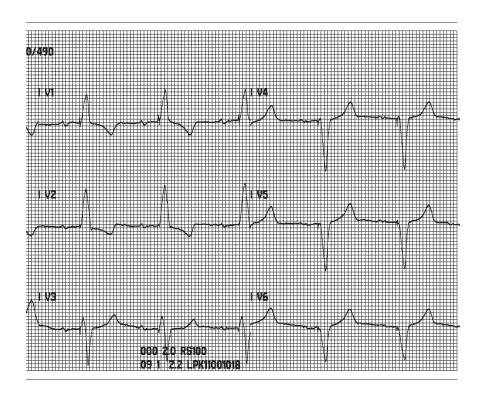
Step 2a Scan each of the leads for T wave inversion (**ischemia**). On the chart below, mark an X in the leads where T wave inversion occurs.

Step 2b Scan each lead for ST segment depression (**ischemia**) and mark the chart.

Step 2c Scan for ST segment elevation (injury), mark the chart.

Step 2d Scan for abnormal Q waves (infarct), mark the chart.

	Т	Ш	III	aVR	aVL	aVF	V1	V2	V3	V4	V5	V6
T wave inversion (T↓)												
ST segment depression (ST↓)												
ST segment elevation (ST1)												
Abnormal Q waves (abn Q)												



Step 3 Localize the ECG changes to an area of the heart. Indicate type of change (i.e. $T\downarrow$, $ST\downarrow$, $ST\uparrow$, abn Q)

_____ Septal (V1, V2) _____ Lateral (I, aVL, V5, V6)

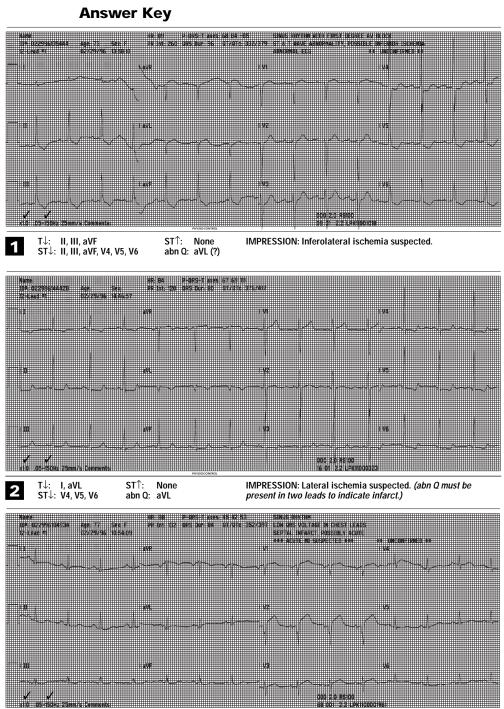
_____ Anterior (V3, V4) ______ Inferior (II, III, aVF)

Step 4 Consider other conditions that can result in ST segment elevation. If no ST elevation is present, skip to Step 5.

- LBBB (Hint: QRS duration ≥ 120ms and complex is negative in Lead V1. Can't make AMI diagnosis by ECG.)
 Electronic ventricular pacemaker rhythm
 Pericarditis
- _____ Left ventricular hypertrophy
- _____ Early repolarization

Step 5 State your impressions and request confirmation/consultation with a physician experienced in 12-lead ECG interpretation. Include the type of condition suspected and the area of the heart affected.

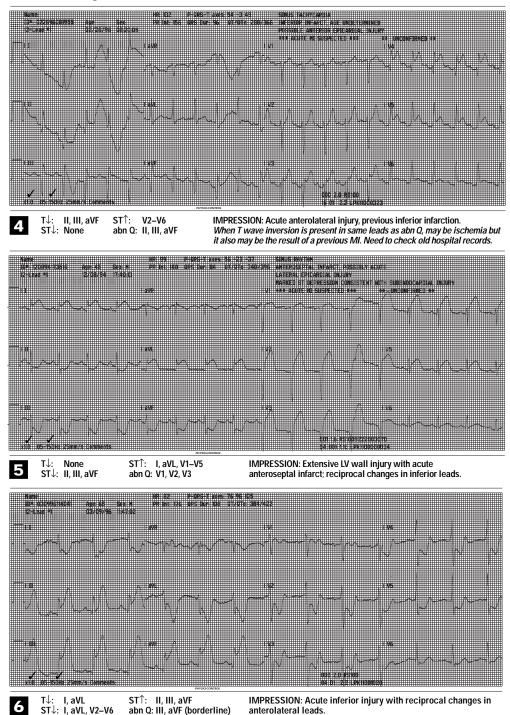
normal ECG	previous infarct
ischemia	condition that mimics
acute injury	acute MI on ECG
acute infarct	

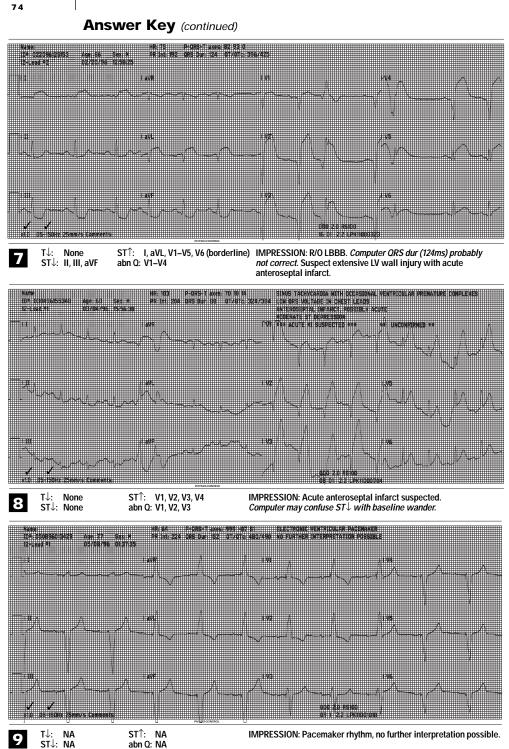


3 T↓: None ST↑: V1, V2, V3 ST↓: None abn Q: V2

IMPRESSION: Acute septal injury/infarction suspected.

Answer Key (continued)





77 Procedure for Obtaining a Multilead ECG Using a 3-Lead ECG Device



Procedure for Obtaining a Multilead ECG Using a 3-Lead ECG Device

- 1) Apply limb lead electrodes to the patient in the RA, LA, and LL positions.
 - Proper skin preparation will improve signal quality.
 - Apply the electrodes to the extremities rather than to the torso when doing a multilead ECG.
- 2) Select the diagnostic frequency response printing option. If using the LIFEPAK[®] 10 defibrillator/monitor, press and hold the RECORD key until the word DIAG appears on the screen.
 - Devices that do not offer the DIAG frequency response option should not be used for the acquisition of multilead ECGs.
- 3) Encourage your patient to lie quietly with arms and legs resting on a supportive surface during the acquisition of the 12-lead.
- 4) Using the LEAD SELECT feature, obtain three to six seconds of ECG trace in Lead I, Lead II and Lead III. Mark each segment with the appropriate lead label. Leave the displayed lead set to Lead III.
- 5) Remove the LL leadwire from the electrode and connect a fresh electrode.
- 6) Find the V1 position (fourth intercostal space, right sternal border) and apply the LL electrode.
- 7) Obtain three to six seconds of ECG trace. Label the lead on the strip.
- Repeat steps 5, 6 and 7 for each of the remaining precordial leads—V2, V3, V4, V5 and V6. Refer to instructions in Chapter 1 on lead placement if necessary.

The procedure for multilead ECG is complete. Note that leads aVR, aVL and aVF are not available using this method. However, you can gain considerably more information from this ECG than you can from a rhythm strip of Lead II. Practicing the technique develops comfort and efficiency in performing a multilead ECG whenever it is indicated.

Sample Protocols

- 81 Indian River County EMS
- 85 Mayfield Village Fire Department/ Meridia Hillcrest Hospital
- 87 Richmond Ambulance Authority
- 91 Tucson Fire Department/University of Arizona
- 94 University of Texas Southwestern Medical Center

The protocols printed in this section are provided courtesy of the EMS systems listed above. They may be useful in developing protocols for your institution. Always follow the protocols authorized by your medical director.

Indian River County

Expanded Chest Pain Guidelines

- 1. Chest Pain
 - A. Administer oxygen per the patient's needs.
 - B. Obtain pertinent chest pain history:
 - O: Onset of Signs/Symptoms
 - P: Provocation of pain
 - Q: Quality of pain
 - R: Radiation of pain
 - S: Severity of pain
 - T: Time of duration of symptoms
 - C. Begin assessment and treatment. Monitor cardiac rhythm and document six second strip. Treat rhythm disturbances per the appropriate guidelines.
 - D. Obtain Standard 3-Lead ECG tracing. If you suspect infarction or injury (by history, clinical presentation and/or ECG), obtain complete 12-Lead ECG and treat according to your assessment (see below).
 - E. If Inferior infarct is suspected (ST segment elevation in Lead II, Lead III and aVF):
 - (1) Establish an IV, and administer 2 grains ASA.
 - (2) Look for ECG and clinical signs of **Right Ventricular** involvement.
 - (a) If present, **do not** administer nitroglycerin or morphine without physician order.
 - (b) If right ventricular infarct is not suspected, proceed with nitroglycerin and morphine, as detailed in section F.2 and F.3 of this guideline.
 - (3) If patient hypotensive, contact ER Physician and request judicious fluid bolus, in 250cc increments, up to 2000cc. Reassess lung sounds after each 250cc increment.
 - (4) Be prepared for the development of AV blocks and bradycardia (usually narrow complex that responds to atropine).

(continued)

Expar	ndec	Chest Pain (continued)					
F.	F. If Anterior Infarct is suspected (ST segment elevation in V1–V6, Le aVL):						
	(1)	Establish an IV and administer two grains ASA.					
	(2)	Administer 0.4mg SL NITROGLYCERIN, every three to five min, up to three doses.					
		(a) Consider use of nitroglycerin 16.5mcg/min IV infusion. May be increased by 5 to 10mcg/min (2 to 3gtts) until desired effect is achieved or systolic blood pressure < 100mmg.					
	(3)	If patient is still experiencing pain, administer 2mg morphine sulfate SLOW IV push. May administer an additional 2mg MS SLOW IV push, prn pain.					
	(4)	Monitor for signs of cardiogenic shock.					
	(5)	Observe for development of new onset bundle branch block and wide complex AV blocks. If present, follow pacemaker guidelines.					
	(6)	If patient is hypotensive , assess lung sounds and place patient in Trendelenburg position as appropriate. Infuse NaCl 200cc as appropriate. priate.					
	(7)	If still hypotensive, may administer DOPAMINE 5ug/kg/min. Titrate up to systolic BP of 90mmHg.					
G.	lf E	CG is Non-Diagnostic or equivocal:					
	(1)	0.4mg nitroglycerin sublingual, q3 to 5 min, up to three doses.					
	(2)	If patient still experiences pain administer:					
		(a) Morphine, 2mg, SLOW IVP, may repeat x 1 prn pain.					
		(b) ASA 2 grains.					
Н.	lf P	rinzmetal's angina is suspected:					
	(1)	document serial changes in 12-lead ECGs, and note times.					
	(2)	correlate ECGs with changes in pain perception and therapeutic actions.					

Indian River County

Thrombolytic Guidelines

- 1. Initial treatment as per CHEST PAIN Guidelines.
- 2. Complete thrombolytic criteria form (shown below) if MI is suspected by history and ECG changes.
 - A. If the patient does not meet all the critical criteria for thrombolytic therapy, continue to manage the patient as per chest pain guidelines.
 - B. If patient does meet the thrombolytic critical criteria:
 - (1) Advise Emergency Department as soon as feasible.
 - (2) Start second IV with double lumen catheter (note any unsuccessful attempts).

THROMBOLYTIC CHECKLIST

Onset of chest discomfort		HRS
Location of suspected infarct		HRS
ST segment elevation	LEAD	MM
	LEAD	MM
	LEAD	MM

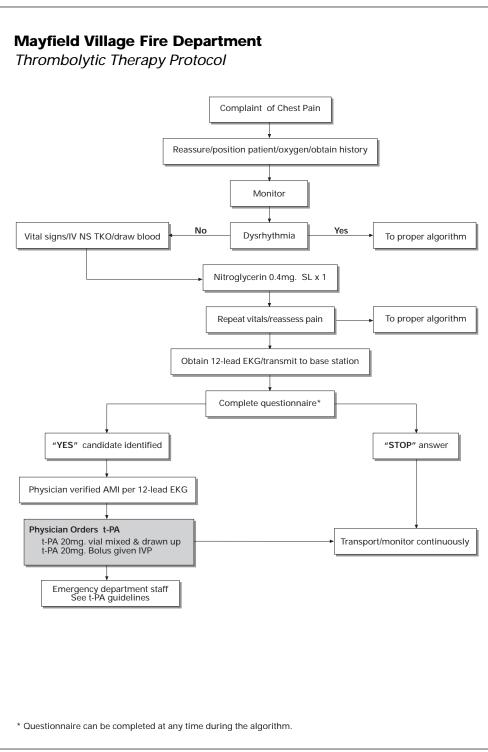
CRITICAL CRITERIA

YES		NO
	Oriented, can cooperate	
	Ongoing heart pain	
BP right ar	m	
BP left arm	·	
	Systolic is more than 80 and less than 180	
	Systolic right arm vs. left arm is less than 20	
	Diastolic is less than 120	
	Active bleeding	
	Criteria met	
(continued)		

Thrombolytic Guidelines (continued)

ADDITIONAL INFORMATION	DATE
Stroke, seizures, brain surgery	
Central lines or trauma	
Takes warfarin (Coumadin)	
Known bleeding problems	
GI bleed in last twelve months (dark/tarry stool)	
Surgery in last two months	
Cancer, terminal	
Kidney, liver problems, diabetes	
Colitis, Crohn's enteritis	
Recent falls	
Notified Hospital	HRS

ASA 2 grains (chewable)	HRS
Number of IVs Established	HRS
Location of any Unsuccessful IV Attempts:	

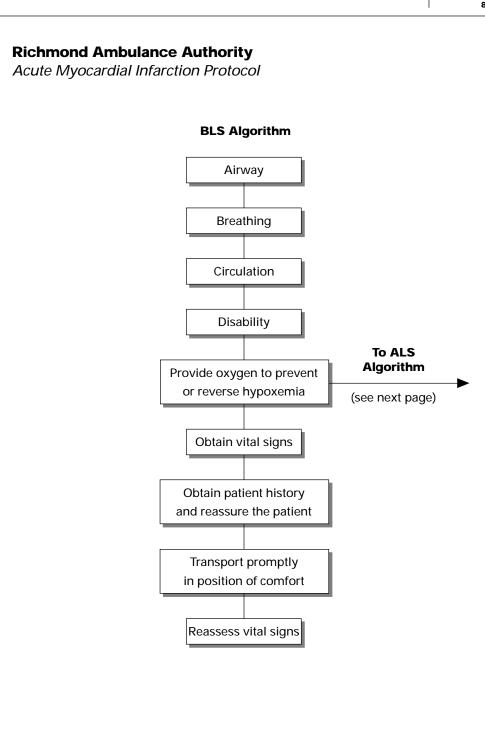


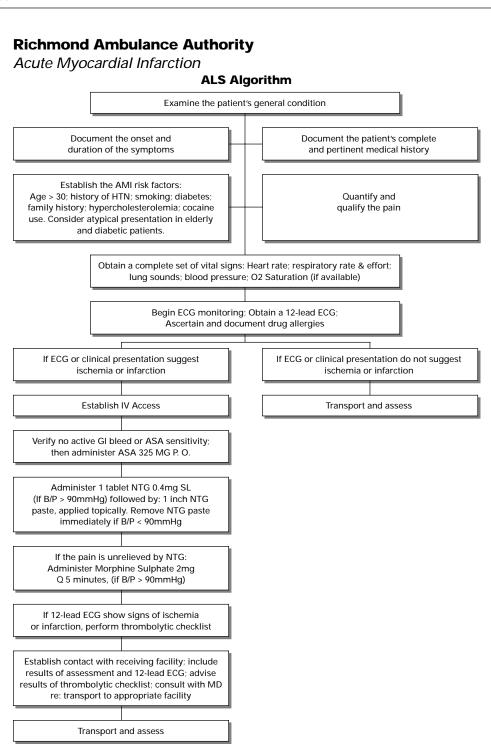
Mayfield Village Fire Department

Thrombolytic Protocol—Inclusion/Exclusion Checklist

Patient name	Date	
EXCLUSION QUESTIONS		
1. History of bleeding disorder?	No	Yes
2. GI or GU bleeding within four weeks?	No	Yes
3. History of stroke?	No	Yes
4. Surgery or organ biopsy in past two weeks?	No	Yes
5. Uncontrolled high B/P? (SPB > 180, DBP > 110)	No	Yes
6. CPR > ten min. last two weeks?	No	Yes
7. Severe trauma in past two months?	No	Yes
8. Cardiogenic shock? (SBP < 80 after CPR)	No	Yes
9. History of diabetic hemorrhagic retinopathy?	No	Yes
If "YES" answer to any of above—STOP—not a candidate.		
INCLUSION QUESTIONS		
 CHEST PAIN < six hours duration? (If > six hours must be severe & continuous) 	Yes Yes	No No
2. AGE—within accepted standards (< 90 years)	Yes	No
3. SEX—female beyond childbearing age?	Yes	No
4. 12-LEAD EKG transmitted & read as MI by physician?	Yes	No
If "NO" answer to any of above—STOP—Not a candidate.		

Paramedic signature





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			89
Richmond Ambulance Authority Acute AMI Thrombolytic Checklist			
B/P Left Arm:/ B/P Right Arm: _	/		
Pain lasting > 15 min < 12 hours	YES	NO	
Systolic B/P > 180	YES	NO	
Diastolic B/P < 120	YES	NO	
> 15mmHg between right arm systolic B/P and left arm systolic B/P?	NO	YES	
History of CNS pathology? (stroke, seizures, brain tumor, neurosurgery)	NO	YES	
Major trauma, surgery (including laser eye surgery, GI or GU bleed) within six weeks?	NO	YES	
Bleeding or clotting problems, or on blood thinners (Coumadin)?	NO	YES	
CPR for > ten minutes?	NO	YES	
Female and pregnant?	NO	YES	
Serious systemic disease? (such as severe kidney or liver disease)	NO	YES	
Advanced or terminal cancer?	NO	YES	

Any answers that fall within the gray box should be brought to the attention of ER staff ASAP.

Richmond Ambulance Authority

Criteria for Thrombolysis in Acute Myocardial Infarction (AMI)

- Chest pain of \geq 15 minutes and \leq 12 hours in duration that is consistent with AMI
- Electrocardiographic changes:
 - ST segment elevation > 0.1mV in at least two continuous leads
 - New or presumably new left bundle branch block (LBBB)
 - Prominent R waves with ST segment depression in V1 and/or V2

University of Arizona Department of Emergency Medicine

Prehospital 12-Lead ECGs: Who, When, Where?

WHO?

- Prehospital 12-lead ECGs are to be acquired on the following patients when the care of the patient will not be compromised by taking the approximately one minute to perform it.
 - 1. Patients calling 9-1-1 with a complaint of acute, non-traumatic, cardiac chest pain.
 - 2. A patient that the paramedic thinks is suffering an acute cardiac event in the absence of a complaint of chest pain.
 - 3. Any patient for whom a physician requests a Prehospital 12-lead ECG be acquired whether or not a cardiac incident.

WHEN AND WHERE?

Suggested sequence:

- Reassure, place in a position of rest and administer 0, as needed
- Place on cardiac monitor using standard limb lead (4) placement if a 12-lead is anticipated
- Complete patient assessment
 - a. Vital signs (bilateral blood pressure when possible)
 - b. History and medications
 - c. Attach precordial electrodes
 - d. Acquire Prehospital 12-lead ECG BEFORE nitroglycerin, aspirin, lidocaine, morphine or other medication whenever reasonable and possible.
 - e.** Continue with ACLS approved protocol for incident type and make base hospital contact if not previously done.
- Remember, if precordial electrodes are attached and 12-lead ECG is acquired while patient assessment is taking place your scene time is not adversely affected.
- Acquire 12-lead ECG either in patient's residence or incident location prior to moving patient to vehicle in preparation for transport or in vehicle just prior to beginning transport.
- Transmit 12-lead ECG to receiving facility either by landline or cellular telephone while in residence or business or by cellular telephone while en route to receiving facility.

Prehospital 12-Lead ECGs: Who, When, Where? (continued)

- When time allows, acquire a second 12-lead ECG during transport AFTER initiation of IV and/or administration of nitroglycerin, aspirin, lidocaine, morphine or other medication. Pre and post medication 12-leads can give the emergency physician/cardiologist additional useful information.
- If either defibrillation or synchronized cardioversion is necessary, quickly remove the precordial leads necessary to allow for the paddles or defibrillation electrodes and proceed with appropriate protocol.

^{**} NOTE: ACLS protocol and physician orders will supersede the 12-lead ECG acquisition in cases where it is in the best interest of the patient.

Thrombolytic Eligibility Checklist Alarm # Rig # This form is to be completed for all patient's complaining of non-traumatic cardiac

University of Arizona Department of Emergency Medicine

This form is to be completed for all patient's complaining of non-traumatic cardiac chest pain or other presenting symptom(s) of possible acute myocardial infarction. It is standard of care for Tucson Fire Department to acquire and transmit a pre-hospital 12-lead ECG on these patients. This criteria is designed to assist the emergency department physician in determining eligibility for possible thrombolytic treatment when indicated and can be reported through telemetry if desired.

Make sure the patient's name, age, sex, heart rate and home address are completed on the First Care Form. Attach this checklist to the First Care Form and forward to EMS office.

INCLUSION CRITERIA:

1.	Does this patient have ST changes cons with AMI (\geq 1mm of ST elevation in at I limb leads or \geq 2mm ST segment eleva two or more contiguous precordial lead	east two tion in	YES	NO
2.	Has this patient had less than six hours symptoms indicative of AMI? Time of or of chest pain		YES	NO
ΕX	CLUSION CRITERIA:			
3.	Does this patient have any history of stroke or central nervous system structural damage? (i.e. neoplasm, aneurysm, or intracranial surgery) YES NO			NO
4.	Does this patient have any active bleedi		YES	NO
ч. 5.	Has this patient had major surgery or significant			NO
Э.	trauma? YES NO		NO	
6.	Has this patient had a recent non-compressible vascular puncture? YES NO		NO	
7.	Has this patient been treated with streptokinase or other thrombolytic agent in the past			
	6 months?		YES	NO
8.	Bilateral Blood Pressure:	Right Arm _	/	
		Left Arm _	/	
	Any Comments:			

University of Texas Southwestern Medical Center

Cardiac Patient Protocol

DEFINITION:

Patients who are complaining of symptoms believed to be of cardiac origin: chest pain, shortness of breath, dizziness, syncope, weakness, palpitations or irregular pulse.

CONTINUED BASIC LIFE SUPPORT:

- 1. Continuously observe and monitor patient for change.
- 2. Loosen any tight, restrictive clothing.
- 3. Place in a sitting or semi-sitting position unless hypotensive (B/P less than 90).
- 4. Transport to ambulance on stretcher.

ADVANCED LIFE SUPPORT:

- If any of the following are present, go to the appropriate protocol. SUSTAINED or NON-SUSTAINED VENTRICULAR TACHYCARDIA, PSVT, BRADYCARDIA, CONGESTIVE HEART FAILURE or SHOCK. If both BRADYCARDIA AND NON-SUSTAINED V-TACH are present, go to bradyarrhythmia protocol first.
- 6. IV D5W TKO.
- 7. If stable, obtain a 12-lead ECG (if available) on patients when any of the following criteria is present:
 - a. chest pain, non-traumatic in origin
 - b. signs and symptoms of CHF less than one month in duration
 - c. physicians request
 - d. paramedics believe the patient may have a cardiac event
- 8. If chest pain is present, complete chest pain checklist.
- 9. CONTACT BIOTEL
- 10. Aspirin 325mg tablet by mouth unless known allergy to Aspirin or history of GI bleed.
- 11. Nitroglycerin (NTG) 0.4mg sublingual spray if systolic B/P is greater than 100 and sustained V-Tach is not present; may repeat twice in 15 minutes.
- 12. If pain persists after NTG or NTG is contraindicated and systolic B/P is greater than 90, administer Nitronox.

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	City/Incident Number:			
= Check mark indicates condition exists or is met				
 Possible indications for thrombolytics 	 Possible contraindications for thrombolytics 			
1. Oriented, able to cooperate	1. History of stroke or brain surgery			
 2. Systolic BP difference < 20 BP right arm / BP left arm / 	2. Acute trauma of any kind			
3. Diastolic BP < 120	 3. Anticoagulant medications (coumadin, warfarin, heparin) 			
4. Systolic BP > 80 and < 180	4. Known bleeding problems			
	5. Any GI bleeding in last 12 month			
	6. Any surgery in last two months			
	7. Terminal cancer			
	8. Significant liver or kidney disease			
	9. Diabetes with visual problems			
	10. Colon inflammation or colon disease, or Crohn's disease			
	11. Current central line (transfer pt.)			

References

- 1. American Heart Association: Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care, Part III—Adult Advanced Cardiac Life Support. *Journal of the American Medical Association.* 1992;268(13):2199–2241.
- 2. American Heart Association: Fact Sheet on Heart Attack, Stroke and Risk Factors—1995 Cardiovascular Statistics. Publ. 51–1089.
- Gruppo Italiano per lo Studio della Streptochinasi nell'Infarcto Miocardico (GISSI): Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. *Lancet.* 1986;1:397–401.
- Weaver WD, Cerqueira M, Hallstrom AP, et al: for the Myocardial Infarction Triage and Intervention Project Group [MITI]. Prehospital-initiated vs hospitalinitiated thrombolytic therapy: The Myocardial Infarction Triage and Intervention Trial. JAMA. 1993;270:1211–1216.
- 5. Pepine CJ: The importance of timing of thrombolytic therapy as shown in clinical trials. *Clin Cardiol.* 1990;13(VIII–12–17).
- Dracup K, Moser DK: Treatment-seeking behavior among those with signs and symptoms of acute myocardial infarction. *Heart & Lung.* 1991;20(5, part 2):570– 575.
- 7. Ho MT: Delays in the treatment of acute myocardial infarction: An overview. *Heart & Lung.* 1991;20(5):566–570.
- Sharkey SW, Brunette DD, Ruiz E, et al: An analysis of time delays preceding thrombolysis for acute myocardial infarction. *J Am Med Assoc.* 1989;262:3171– 3174.
- National Heart Attack Alert Program [NHAAP] Coordinating Committee, 60 Minutes to Treatment Working Group: Emergency department: rapid identification and treatment of patients with acute myocardial infarction. *Annals Emerg Med.* 1994;23(2):311–329.
- Gibler WB, Kereiakes DJ, Dean EN, et al: Prehospital diagnosis and treatment of acute myocardial infarction: A North-South perspective. *Am Heart J*. 1991;121(1, part 1);1–11.
- Karagounis L, Ipsen SK, Jessop MR, et al: Impact of field-transmitted electrocardiography on time to in-hospital thrombolytic therapy in acute myocardial infarction. *Am J Cardiol.* 1990;66:786–791.
- Keriakes DJ, Weaver WD, Anderson JL, et al: Time delays in the diagnosis and treatment of acute myocardial infarction: A tale of eight cities. Report from the Pre-hospital Study Group and the Cincinnati Heart Project. *Am Heart J*. 1990;120(4):773–780.
- Grim PS, Felman T, Childers RW: Evaluation of patients for the need of thrombolytic therapy in the prehospital setting. *Annals Emerg Med.* 1989;18(5):483– 488.

- Aufderheide TP, Hendley GE, Thakur RK, et al. The diagnostic impact of prehospital 12-lead electrocardiography. *Annals Emerg Med.* 1990;19(11):1280–1287.
- Hanisch, P: Identification and treatment of acute myocardial infarction by electrocardiographic site classification. *Focus on Critical Care*. 1991;18(12):480–488.
- 16. Connor R: Coronary artery anatomy: the electrocardiographic and clinical correlations. *Crit Care Nurse*. 1983;3:68–73.
- Zalenski, R, Cooke D, et al: Assessing the diagnostic value of an ECG Containing Leads V4R, V8 and V9: the 15-lead ECG. *Annals Emerg Med.* 1993;22(5):786–793.
- Stewart, S, Haste, M: Predictions of right ventricular and posterior wall ST elevation by coronary care nurses: The 12-lead electrocardiograph versus the 18-lead electrocardiograph. *Heart and Lung.* 1996;25:14–23.

Bibliography

- Davis D: How to Quickly and Accurately Master ECG Interpretation. Philadelphia, JB Lippincott, 1985.
- Fassler, MD, Steuble, BT: Electrocardiogram Interpretation and Emergency Intervention, Springhouse Corp., 1991.
- Gibler, WB, Aufderheide, TP: Emergency Cardiac Care, St. Louis, Mosby—Year Book, Inc., 1994.
- Grauer, K: A Practical Guide to ECG Interpretation, St. Louis, Mosby—Year Book, Inc., 1992.
- Huzar, RJ: Basic Dysrhythmias, 2nd ed. St. Louis, Mosby—Year Book, Inc., 1994.
- Phalen, T: The 12-Lead ECG in Acute Myocardial Infarction. St. Louis, Mosby Lifeline, 1996.
- Schamroth L: The 12-Lead Electrocardiogram, Book 1. Chicago, Year Book Medical, 1989.
- Thaler MS: The Only EKG Book You'll Ever Need. Philadephia, JB Lippincott, 1988.
- Wellens JJ, Conover MJ: The ECG in Emergency Decision Making. Philadelphia, WB Saunders, 1992.



Glossary

AMI

Acute myocardial infarction.

anatomically contiguous leads

Any two or more ECG leads that view the same aspect of the heart, e.g., Leads I and aVL look at the lateral wall of the left ventricle.

angle of Louis

The point where the manubrium joins the sternum; used as a landmark to guide the proper application of precordial ECG electrodes.

anterior axillary line

An imaginary line extending from the front of the axilla (armpit) toward the waist; used as a landmark to guide the proper application of precordial ECG electrodes.

artifact

Distortion of the ECG trace due to interference with the cardiac signal.

biphasic

A waveform that has two phases, i.e., a QRS complex that falls both above and below the isoelectric line.

bipolar lead

Composed of two electrodes: one positive pole and one negative pole; Leads I, II and III are bipolar.

circumflex coronary artery (Cx)

A branch of the left coronary artery that typically supplies blood to the left atrium and the posterior and lateral walls of the left ventricle.

conduction

The passage of electrical impulses along specialized nerve tissue of the heart.

depolarization

Process by which cells of the heart are electrically activated; necessary for muscle contraction to occur.

diagnostic frequency response

A mode for ECG recording in a wide signal bandwidth; the standard for 12-lead ECG devices is 0.05 to 150Hz.

diagnostic quality ECG

A 12-lead ECG acquired and printed in diagnostic frequency response.

electrocardiogram

The graphic representation of the heart's electrical activity.

equiphasic

A biphasic waveform in which the portion lying above the isoelectric line is equal in amplitude to the portion lying below the isoelectric line.

frequency response

A setting on the device that defines the range of ECG signals that can be authentically reproduced on the screen or the recorder of an ECG monitor.

frontal plane

Orientation of the heart's electrical activity as observed from the limb leads; provides information about rightward, leftward, inferior and superior current flow.

horizontal plane

Orientation of the heart's electrical activity as observed from the chest leads; provides information about anterior, anterolateral and anteroseptal and posterior current flow.

intercostal space

The hollow space between the ribs.

ischemia

A condition in which there is inadequate oxygenation of tissue.

isoelectric line

The parts of the ECG trace in which there are no positive or negative deflections; the baseline.

infarction (acute myocardial)

A condition in which heart muscle has died; *acute* implies that some heart muscle at risk of dying can be rescued with prompt treatment.

injury (acute myocardial)

A condition which occurs when ischemia results in cell damage; may be reversible or may proceed to cell death.

left anterior descending coronary artery (LAD)

A branch of the left coronary artery which supplies blood to the anterior wall of the left ventricle, the interventricular septum, and the bundle of His and bundle branches.

J point

Point on the ECG trace where the S wave transitions to the ST segment; used for the determination of ST segment elevation or depression.

limb leads

ECG leads I, II, III, aVR, aVL and aVF; record the frontal plane.

manubrium

A thickened bony structure at the juncture of the second rib and the sternum.

mean vector

The net direction of electrical activity when all vectors are combined.

midclavicular line

An imaginary line extending from the midpoint of the clavicle; used to find the proper location for the lead V4 electrode.

midaxillary

An imaginary line extending from the middle of the axilla (armpit); used to find the proper location for the lead V6 electrode.

monitor frequency response

A mode for ECG recording in a narrow signal bandwidth; typically 1 to 30Hz or 0.5 to 40Hz.

National Heart Attack Alert Program (NHAAP)

A multidisciplinary committee of health care professionals who work to promote early detection and early access to care for patients with heart attack; part of the National Institutes of Health.

percutaneous transluminal coronary angioplasty (PTCA)

An invasive procedure which aims to reestablish blood flow in a occluded blood vessel of the heart.

PR interval

Component of the ECG trace measured from the beginning of the P wave to the beginning of the QRS complex.

precordial electrodes

Chest electrodes placed to record leads V1 through V6.

precordial leads

Leads V1, V2, V3, V4, V5 and V6; record electrical activity of the heart in the horizontal plane.

Q wave

First negative deflection of the QRS complex.

QRS complex

Component of the ECG trace representing ventricular depolarization; comprised of Q, R and S deflections (all three may not always be present).

QRS duration

Component of the ECG trace measured from the beginning of the Q wave to the beginning of the ST segment.

reciprocal ECG changes

Indirect ECG changes that present a mirror image of the direct ECG changes observed in an area of the heart.

repolarization

Process by which depolarized cells return to their normal resting state.

right coronary artery (RCA)

The artery that typically supplies blood to the right atrium, the SA and AV nodes, and the posterior wall of the left ventricle.

right ventricular infarction

Death of heart muscle occurring in the right ventricle; occurs in conjunction with approximately 40% of inferior wall MIs.

sensitivity

The ability to detect a condition when it is present.

specificity

The ability to rule out a condition when it is not present.

ST segment

Component of the ECG trace between the end of the QRS complex and the T wave; normally isoelectric.

sternum

The breastbone; anchors the ribs anteriorly.

T wave

Component of the QRS complex representing ventricular repolarization; deflection that follows the ST segment.

TP segment

Component of the ECG trace measured from the end of the T wave of one ECG cycle to the beginning of the P wave of the next ECG cycle.

thrombolytic therapy

A drug therapy which aims to restore blood flow in an artery by dissolving an occluding blood clot.

12-lead ECG

Recording of the heart's electrical activity using six limb leads and six precordial leads.

unipolar leads

Composed of one positive electrode and a common negative reference point; leads aVR, aVL, aVF, V1–V6.

vector

Direction of electrical current flowing through the heart.

voltage

A measurement of electrical potential differences occurring at the various electrode sites.

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