

Basic Electrocardiographic Techniques

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The electrocardiogram (ECG) is a graphic recording of the electrical activity of the heart. The standard ECG is obtained by applying electrodes over the chest and limbs that record the electrical activity of the cardiac cycle. Developed nearly 100 years ago, the ECG remains the most important initial diagnostic tool for the assessment of myocardial disease, ischemia, and cardiac dysrhythmias.

Electrocardiography is performed widely throughout the health care field, including ambulances, ambulatory clinics, emergency departments (EDs), and in-patient hospital units. Standard electrocardiography machines are small, self-contained, and portable, allowing them to be employed in virtually any setting. As a result, clinicians, nurses, and many other health care providers should be familiar with the procedure of standard 12-lead electrocardiography. Emergency clinicians should also be familiar with the alternative leads and other accessory techniques available in electrocardiography, as well as the pitfalls of lead misplacement, misconnection, and tracing artifacts.



BACKGROUND CAN BE FOUND ON EXPERT CONSULT

INDICATIONS

The most frequent indication for electrocardiography performance in the ED is the presence of chest pain. Other common indications include abnormal rhythm, palpitations, dyspnea, syncope, diagnosis-based (e.g., acute coronary syndrome [ACS], suspected pulmonary embolism), and system-related (e.g., “rule-out myocardial infarction [MI]” protocol, admission purposes, and operative clearance) indications.⁹ The ECG is used to help establish a diagnosis, select appropriate therapy, determine the response to treatment, assist in the correct disposition for the patient, and help predict risk of both cardiovascular complication and death.

The initial 12-lead ECG obtained in the ED can be an important tool for determination of cardiovascular risk and, as such, the choice of in-hospital admission location. Brush and coworkers¹⁰ classified the initial ECG into high- and low-risk groups. The low-risk electrocardiographic group had normal ECGs, nonspecific ST-T-wave changes, or no change when compared with a previous ECG. High-risk ECGs had significant abnormalities or confounding patterns—such as pathologic Q-waves, ischemic ST segment or T-wave changes, left ventricular hypertrophy, left bundle branch block, or ventricular paced rhythms. Patients with initial ECGs classified as low risk had a 14% incidence of acute myocardial infarction

(AMI), 0.6% incidence of life-threatening complications, and a 0% mortality rate. Patients with initial ECGs classified as high risk had a 42% incidence of AMI, 14% life-threatening complications, and 10% mortality rate.¹⁰ Another approach to risk prediction involves a simple calculation of the number of electrocardiographic leads with ST segment deviation (elevation or depression)—with an increasing number of leads being associated with higher risk. Along similar lines, the clinician is also able to predict risk with a summation of the total millivolts of ST segment deviation; once again, higher totals are associated with greater risk.¹⁰

The limitations of the ECG must be recognized, however. The ECG is widely reported to have a sensitivity for AMI of only approximately 55%; in one study of 1000 patients with ischemic symptoms, that sensitivity improved to 68% with serial ECGs and ST segment trend monitoring.¹¹ In another series, the sensitivity of the ECG for AMI ranged from 43% to 65% over a 12-hour period after ischemic symptom onset, yet the negative predictive value of a normal ECG (defined as normal or with nonspecific changes or isolated fascicular blocks) for AMI did not improve above 93% during this period.¹² In a large series of over 10,000 patients of whom 889 (8%) were ultimately diagnosed with AMI, 19 (2%) were inappropriately discharged from the ED. A nonischemic ECG emerged as one of five risk factors for that inappropriate disposition decision (along with female, <55 yr old, nonwhite race, and dyspnea as a chief complaint); 2 of those 19 had a normal tracing, and the other 17 had nonischemic findings on their ECGs.¹³

BASIC EQUIPMENT

The 12-Lead ECG

Although there is variability depending upon the workplace, most ECGs in use today are three-channel recorders with computer memory. Such multichannel systems, recording electrical events in several leads concurrently, offer advantages over the antiquated single-channel recorder systems—capturing transient events on multiple leads simultaneously; banking the data in computer memory for storage, comparison, and transmission; and allowing for data presentation on a single sheet of paper.¹⁴ The electrocardiographic tracing is printed in a standardized manner on a standardized paper by the electrocardiograph, which has default settings regarding the speed with which the paper moves through the machine as well as the amplitude of the deflections to be made on the tracing. Electrocardiographic paper is divided into a grid, with a series of horizontal and vertical lines; the thin lines are 1 mm apart, and the thick lines are separated by 5 mm. At the standard paper speed of 25 mm/sec, each vertical thin line thus represents 0.04 sec (or 40 msec), and the thick vertical lines correspond to 0.20 sec (or 200 msec). Recordings from each of the 12 leads typically are displayed for 2.5 seconds by default setting; the leads appearing horizontally adjacent to each other are separated by a small vertical hash mark to represent lead change.

The standard ECG includes 12 leads derived from 10 electrodes placed on the patient; each is color-coded and represented by a two-character abbreviation (Table 14-1). The placement of limb leads on the left and right arms (LA and RA, respectively) and the left and right legs (LL and RL, respectively) by color can be recalled with the help of several mnemonics. These include the following: “Christmas trees

TABLE 14–1 Conventional Leads for the 12-Lead Electrocardiogram

Location	Notation	Color
Right arm	RA	White
Left arm	LA	Black
Left leg	LL	Red
Right leg	RL	Green
Precordial leads	V ₁	Brown/red
	V ₂	Brown/yellow
	V ₃	Brown/green
	V ₄	Brown/blue
	V ₅	Brown/orange
	V ₆	Brown/violet

below the knees (the green and red leads are placed on the lower extremities); “white on right and green to go” (the white lead is placed on the RA, and the green lead is placed on the leg that controls the gas pedal, while the red lead is correspondingly placed on the leg that is closer to the brake); and “smoke over fire” (the black LA lead is placed over the red LL lead, as with telemetric monitoring pads). Use of these mnemonics may help prevent right/left confusion during electrode placement—and the consequences of limb electrode reversal and misinterpretation of the ECG (see “[Electrode Misplacement and Misconnection](#),” later in this chapter).

Standard 12 Leads

The standard 12-lead ECG depicts cardiac electrical activity from 12 points of view, or leads, that can be grouped according to planar orientation. Six leads (I, II, III, aVR, aVL, and aVF) are oriented in the frontal, or coronal, plane and derived from the four limb electrodes. The six precordial leads (V₁, V₂, V₃, V₄, V₅, and V₆) are oriented in the horizontal, or transverse, plane, with each representing cardiac electrical activity from that perspective. Leads I, II, and III are termed *limb leads*, and are bipolar in that they record the potential difference between two electrodes (Fig. 14–1). The fourth electrode located on the right leg serves as an electrical ground. The positive poles of these bipolar leads lie to the left and inferiorly, approximating the major vector forces of the normal heart. This early convention was established so that the tracing would feature primarily upright complexes. In contrast, augmented leads aVR, aVL, and aVF are unipolar leads, with the positive electrode located at the respective extremities. These augmented leads serve to fill the electrical gaps between the leads I, II, and III. Lead aVR stands alone with a polarity and resultant orientation opposite to the other limb and augmented leads, owing to the fact that its positive electrode is located in the opposite direction (superior and to the right) of the major vector force of the normal heart (inferior and to the left); thus its complexes usually appear “opposite” to those in most or all of the other leads.

Merging of the vector axes of the limb and augmented leads around a central axis yields a hexaxial system representing cardiac electrical activity in the frontal plane (Fig. 14–2). The six precordial leads, oriented in the horizontal plane, represent six unipolar electrodes with vector positivity oriented toward the chest surface, with the central terminal of the hexaxial system serving as a negative pole. In contrast to the frontal plane leads, the angles between each of the pre-

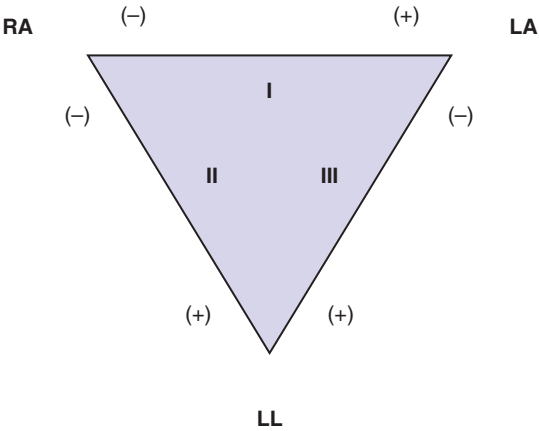


Figure 14–1 Bipolar limb leads. Leads I, II, and III are shown as a triangle, known as *Einthoven's triangle*. Left arm (LA), right arm (RA), and left leg (LL) placement is shown. These bipolar leads are oriented such that the positive poles lie inferiorly and to the left (given that the bottom apex of the triangle is directed toward the left leg), as does the major electrical vector of the heart.

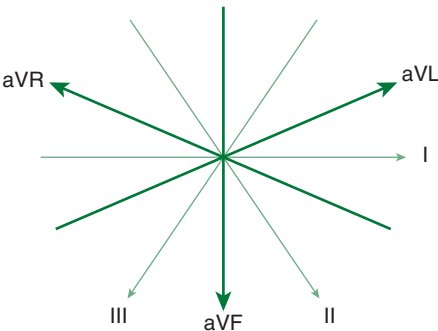


Figure 14–2 Hexaxial system of limb and augmented leads in the frontal plane. Each lead is separated by 30° in this frontal plane representation of the limb and augmented leads. Augmented leads are shown in **boldface**. Arrows denote positive polarity. Note that the inferior leads (II, III, aVF) logically lie at the bottom of this figure, and the lateral leads (I, aVL) lie on the left side of the figure, where the lateral aspect of the heart is located if this were superimposed on a patient.

cordial leads in the horizontal plane are not equal, however. They can vary depending upon electrode placement and body habitus.

Electrode Placement

The four limb electrodes are conventionally placed on the extremities as follows: RA on the right wrist; LA on the left wrist; RL on the right ankle; and LL on the left ankle. Electrodes may be affixed more proximally on the limbs if necessary (e.g., amputation, severe injuries), ideally with a notation made on the ECG.¹⁵ Others note that the electrodes may be placed on any part of the arms or legs, providing they are distal to the shoulders or inguinal/gluteal folds, respectively.¹⁶

Mason-Likar electrode placement is commonly used by hospital staff and paramedics; this approach does not alter precordial electrode placement, but moves the limb electrodes to the torso. Originally described in 1966, the Mason-Likar configuration differs from standard electrode placement in that the arm electrodes are relocated to the infraclavicular

fossae (medial to the borders of the deltoid muscles and 2 cm below the clavicles) and the leg electrodes are positioned along the anterior axillary lines (halfway between the costal margins and the iliac crests). Actual torso positioning may differ in practice owing to individual variation or an attempt to simulate limb electrode placement. A rightward frontal plane access shift has been described when torso electrode placement is used for the limb electrodes, instead of standard positioning on the extremities. Mason-Likar positioning also has been associated with diminution of inferior Q-waves, thus making detection of inferior MI more difficult.¹⁷

The precordial electrodes should be placed as follows: V₁—right sternal border, fourth intercostal space; V₂—left sternal border, fourth intercostal space; V₃—midway between V₂ and V₄; V₄—left midclavicular line, fifth intercostal space; V₅—left anterior axillary line, same horizontal level as V₄; and V₆—left midaxillary line, same horizontal level as V₄ and V₅. Note that V₄ to V₆ are placed at the same horizontal level, not all in the fifth intercostal space. If V₅ and V₆ are situated following the contour of the intercostal space rather than on the same horizontal level, they will be superiorly displaced as the ribs curve around the side of the thorax (Fig. 14-3).

Intercostal space number can be determined by first palpating the sternal angle (angle of Louis), which is the junction of the manubrium and body of the sternum. This transverse bony ridge is located about 5 cm caudad from the sternal notch in the adult. Immediately lateral and inferior to it is the second intercostal space; two spaces farther down lies the fourth intercostal space, where V₁ and V₂ should be placed. Alternatively, one can count down from the medial clavicle; beneath the clavicle lies the first rib, below which is the first intercostal space. Precordial electrodes should not be simply “eyeballed” by the technician, because as little as 1 to 2 cm of electrode displacement can result in significant morphologic alteration in the precordial QRS complexes.^{17,18}

If the patient's anatomy or injury precludes placement of a precordial electrode as described previously, it is permissible to attach it within the radius of the width of one interspace of the recommended position, with appropriate notation on the tracing. If the situation demands further displacement, it is recommended that the lead be omitted, with appropriate documentation on the tracing.¹⁵

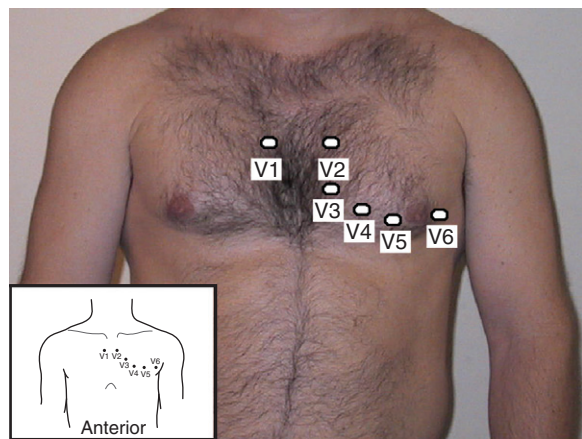


Figure 14-3 Precordial lead placement for the standard 12-lead electrocardiogram (ECG). If multiple or repeat electrocardiographic tracings are anticipated, the original lead placements should be marked on the patient's chest wall or stick-on leads should be left in place after the electrocardiographic wires are removed.

Pediatric Electrode Placement

In addition to the standard 12-lead tracing, leads V₄R and V₃R should also be recorded; these are mirror images of their left-sided counterparts (see “Additional Leads” later in this chapter). The chest of the tiny infant may not accommodate all the precordial electrodes; in such cases, the following array is recommended: V₃R or V₄R, V₁, V₃, and V₆. Limb electrode placement is as in adults.¹⁹

FEATURES OF THE ECG

Discussion of the interpretation of the ECG is beyond the scope of this chapter. Other features of the procedure itself, including a description of the other data found on the electrocardiographic tracing, are detailed later.

Information Provided by the Computer

In addition to the patient demographic data entered by the operator, the tracing will often feature computations regarding rate, intervals, and axes along the top of the paper. On some tracings, a computer-generated “reading” will also be displayed at the top of the tracing. These interpretations are not infallible. A sample of nine of these programs was compared with the readings of eight cardiologists; the “gold standard” in this study was the clinical diagnosis made independently of the interpretations of these tracings, based on other objective data (e.g., echocardiography, cardiac catheterization). The performance of the programs was good, with correct interpretations in a median of 91% of cases, but the cardiologists were significantly better (median 96% correct).²⁰ The computer programs demonstrated a median sensitivity for anterior and inferior MI of 77% and 59%, respectively.²⁰ Of note, this study did not evaluate interpretations of acute ischemia and cardiac rhythm disturbance—perhaps the most critical issues in electrocardiographic interpretation. Others have found both the computer programs and the clinicians to be lacking in their ability to exclude cardiac disease with the ECG, with a negative predictive value for each between 80% and 85%.²¹ The bottom line is this: It is worthwhile to read and consider the computer reading of the ECG, but the emergency clinician should not be beholden to it.

Adjustable Features

Somewhere on the tracing, notation of the electrocardiographic paper speed (in millimeters per second), the calibration (in millimeters per millivolt), and the frequency response (in hertz) will be evident (in Fig. 14-3, these appear in the left lower corner of the tracing). *Calibration*, or standardization, refers to the amplitude of the waveforms on the tracing. It is usually set at a default value of 10 mm/mV, and is graphically depicted by a plateau-shaped waveform that appears at the extreme left side of the tracing, in front of the first complex (Fig. 14-4A). This calibration can be modified by the operator, or by the computer itself, as was the case in Figure 14-4B, in which the patient appeared to have acquired voltage criteria for left ventricular hypertrophy, when in reality, the tracing was unchanged from his baseline (see Fig. 14-4A). Increasing the calibration to 20 mm/mV is helpful when trying to decipher P-wave morphology. Decreasing the calibration to 5 mm/mV is helpful in cases in which the amplitude of the QRS complex (usually in the precordial leads) is so large it



Figure 14-4 A, Normal 10-mm/mV calibration. Note the box-shaped mark to the left of the complexes (arrows); this is a graphic representation of the calibration for the tracing. This parameter should be routinely noted before electrocardiographic interpretation. Note the change in B. B, Abnormal 20-mm/mV calibration. The calibration in this tracing was (inexplicably and unexpectedly) changed to 20 mm/mV by the ECG, not by the operator. When compared with a baseline ECG, it appeared that the patient had developed voltage criteria for left ventricular hypertrophy as well as ST segment elevation. A was recorded minutes later with correction of calibration to the standard 10 mm/mV and was unchanged from baseline tracings.

encroaches upon those of the adjacent leads. Standardization may not be uniform throughout a given tracing. At times, the calibration will be adjusted automatically by the ECG based upon the waveform amplitudes it perceives. For example, it is possible to have normal calibration (10 mm/mV) in the limb and augmented leads, with half-standard calibration in the precordial leads (5 mm/mV); this may occur in instances of marked left ventricular hypertrophy. In this case, the calibration pulse at the left-hand side of the paper will have a downward stairstep appearance.

Paper speed usually is set at a default of 25 mm/sec. It may be manipulated for purposes of deciphering a dysrhythmia, as described later (see “[Alteration in Amplitude and Paper Speed](#)”). It is important that the clinician examine all electrocardiographic tracings for standardization and paper speed parameters before rendering an interpretation.

ADDITIONAL LEADS

Although not considered standard of care in the routine evaluation of patients in the ED, additional electrocardiographic leads may be used in the evaluation of the patient with possible ACS. These additional, or nontraditional, leads include posterior leads (V_7 , V_8 , and V_9), right ventricular leads (especially V_4R), and procedural leads (transvenous pacemaker wire placement and pericardiocentesis). Acute posterior and right ventricular MIs are likely to be underdiagnosed, because the standard 12-lead ECG does not assess these areas directly. The standard ECG coupled with these additional leads con-

stitutes the 15-lead ECG, the most frequently employed extralead ECG in clinical practice.

15-Lead ECG

In a study of all ED chest pain patients, Brady and associates²² reported that the 15-lead ECG provided a more accurate description of myocardial injury in those patients with AMI, yet failed to alter rates of diagnoses or use of reperfusion therapies or to change disposition locations. Looking at a more select population of ED patients, Zalenski and colleagues²³ investigated the use of the 15-lead ECG in chest pain patients with a moderate-to-high pretest probability of AMI, who were already identified as candidates for hospital admission. In this 15-lead ECG study, the authors reported a 12% increase in sensitivity with no loss of specificity (i.e., no increase in false-positive findings) for the diagnosis of ST segment elevation AMI. They concluded that “the findings of ST segment elevation by use of these extra leads can strengthen the ED diagnosis of acute myocardial infarction on the initial tracing and may provide an indication for thrombolytic treatment.” They further suggested that, in the diagnosis of posterior AMI, leads V_8 and V_9 are superior to reliance on detecting the reciprocal ST segment depression seen in leads V_1 to V_3 .

Possible indications for 15-lead ECGs in patients with suspected acute ischemic heart disease include (1) ST segment depression in leads V_1 through V_3 ; (2) all ST segment elevation inferior and lateral AMIs; or (3) isolated ST segment

elevation in leads V_1 and/or V_2 . These indications, despite their apparent clinical utility, remain unproved, and the 15-lead ECG is currently not considered standard of care in the ED.

Posterior Leads

The posterior electrodes V_8 and V_9 are placed on the patient's back— V_8 at the tip of the left scapula and V_9 in an intermediate position between lead V_8 and the left paraspinal muscles. An additional electrode, V_7 , may also be used and is placed on the posterior axillary line equidistant from electrode V_8 (Fig. 14–5). The degree of ST segment elevation in the posterior leads is often less pronounced than the ST segment elevation seen in the standard 12 leads in patients with ST segment elevation AMI. This diminution of posterior lead ST segment elevation results from both the relatively greater distance of these leads from the posterior surface of the heart and the presence of air and soft tissue between the epicardium and the electrocardiographic electrodes.

Right-Sided Leads

The right ventricular electrocardiographic electrodes are placed across the right side of the chest in a mirror image of the standard left-sided electrodes and are labeled V_1R to V_6R ;

alternatively, RV_1 to RV_6 is another commonly used nomenclature for this electrode distribution (Fig. 14–6). Lead V_4R (right fifth intercostal space midclavicular line) is the most useful lead for detecting ST segment elevation associated with right ventricular infarction and may be used solely in the evaluation of possible right ventricular infarction. The ST segment elevation that occurs in association with right ventricular infarction is frequently quite subtle, reflecting the relatively small muscle mass of the right ventricle; at other times, the ST segment elevation is quite prominent, similar in appearance to the ST segment changes seen in the standard 12 leads (Fig. 14–7).

Invasive Procedural Leads

A patient may present with a severely compromising bradydysrhythmia and require a transvenous pacemaker. In such instances, the pacing wire must be placed without the benefit of fluoroscopy. The wire can be advanced using electrocardiographic guidance. Such placement requires that the patient be connected to the limb leads of a grounded electrocardiographic machine and the pacing wire connected to the V lead. As the electrode enters the superior vena cava and high right atrium, the P-wave and QRS complex will be negative. While traversing the atrium, the P-wave and QRS complex will become positive, with the latter becoming larger as the ven-

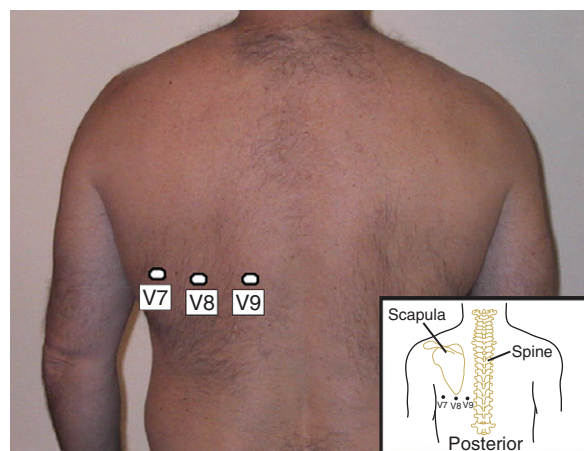


Figure 14–5 Posterior lead placement. Leads V_7 , V_8 , and V_9 are placed on the same horizontal plane as V_6 , with V_7 at the posterior axillary line, V_8 at the tip of the left scapula, and V_9 near the border of the left paraspinal muscles.

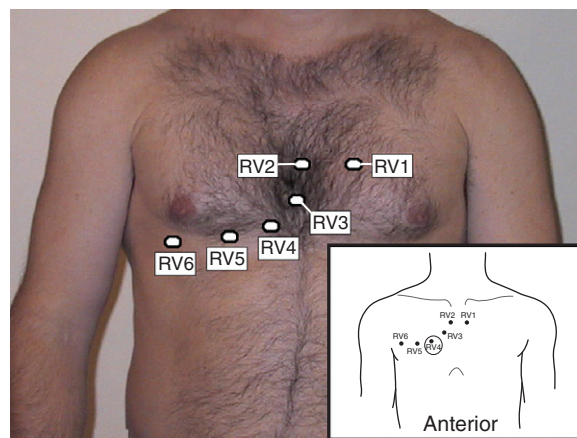


Figure 14–6 Right-sided lead placement. Right-sided leads RV_1 – RV_6 are placed on the chest as a mirror image of the standard precordial leads.

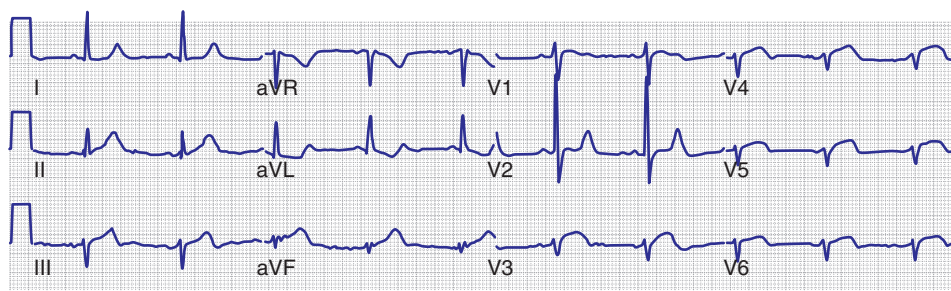


Figure 14–7 Right-sided precordial leads. This tracing displays right-sided precordial leads in an elderly male with chest pain consistent with an acute coronary syndrome. There is ST segment elevation that is somewhat subtle in the inferior leads (II, III, and aVF), which together with the reciprocal ST segment depression seen in lead aVL is consistent with a diagnosis of acute inferior myocardial infarction. Leads V_1 – V_6 are in actuality leads V_1R – V_6R —right-sided precordial leads. The convex upward ST segment elevation seen in leads V_3R – V_6R is indicative of a concomitant right ventricular infarction. This patient had a subtotal proximal occlusive lesion of his right coronary artery at cardiac catheterization.

tricle is approached. If a balloon-tipped flotation catheter is used, the balloon should be deflated once it is in the right ventricle, and advanced until contact is made with the endocardium and the ventricle is captured. Ventricular wall contact is indicated by marked ST segment elevation.

In patients with suspected pericardial effusion who undergo urgent pericardiocentesis, an electrocardiographic lead may be placed on the syringe needle; this form of monitoring assists in the correct positioning of the catheter in the pericardial space. With advancement of the needle, ST segments are monitored. The sudden appearance of ST segment elevation indicates that the needle has moved too far internally (i.e., beyond the pericardial space) and has made contact with the epicardium.

Body Surface Mapping

An emerging electrocardiographic tool, body surface mapping employs numerous leads, thus providing a more detailed electrical description of the heart when compared with the 12-lead ECG. The body map ECG most commonly used in

clinical practice is based upon an 80-lead ECG—64 anterior leads and 16 posterior leads. This more detailed imaging of the myocardium allows for potentially greater diagnostic accuracy in the early detection of ST segment elevation AMI, as well as detection of infarction in more traditionally electrocardiographic “silent” areas of the heart.²⁴⁻²⁶

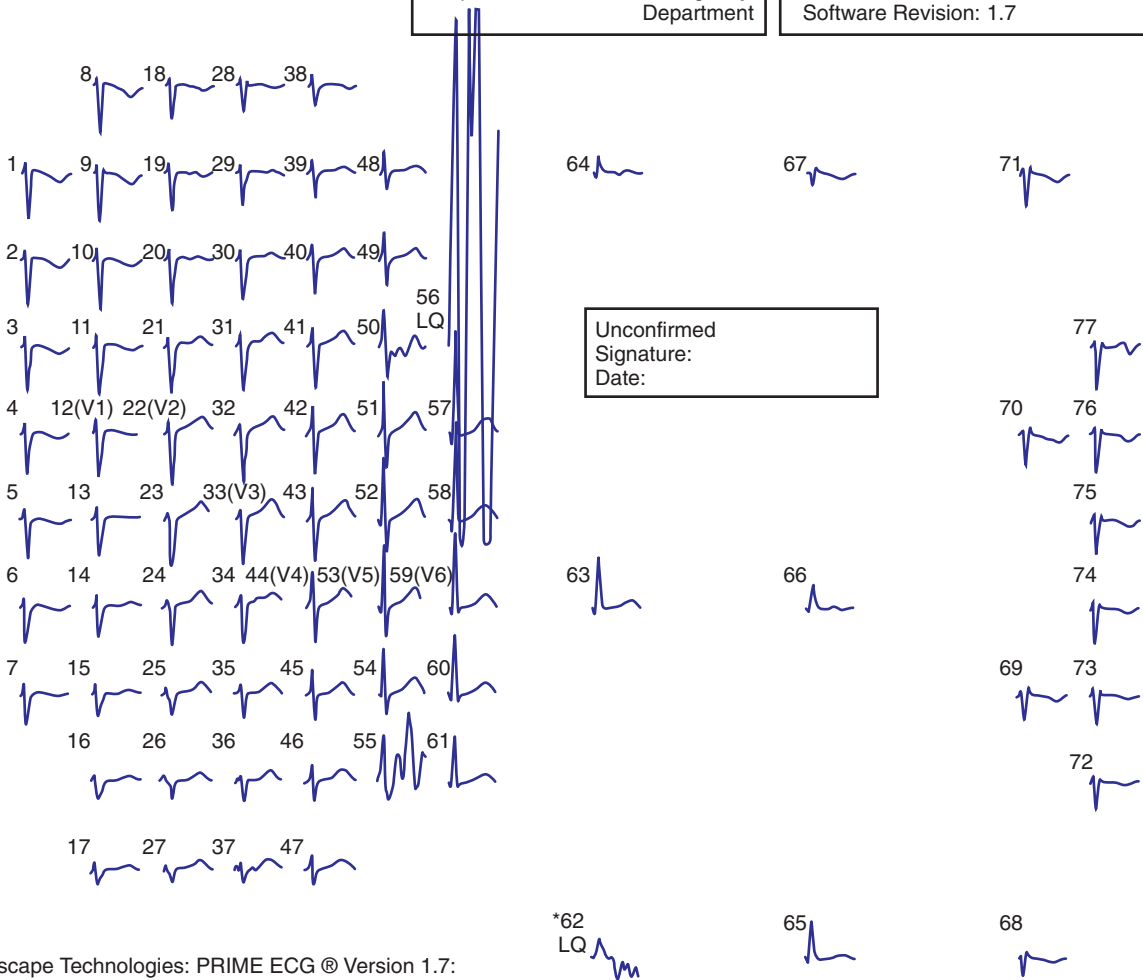
Clinical information is displayed in three basic formats: a 12-lead ECG, an 80-lead ECG (Fig. 14-8), and torso maps (Fig. 14-9). The 12-lead ECG is a standard ECG with 12 leads. It differs only in that, in addition to the standard 12-lead electrocardiographic lead descriptors, the 80-lead numerical lead marker is also displayed when the 12- and 80-lead ECGs share a common lead. For example, leads V₁ and V₂ will also have lead markers 12 and 22, respectively. The 80-lead ECG (see Fig. 14-8) demonstrates a single electrocardiographic P-QRS-T cycle for all 80 leads.

The torso maps are also displayed in four distinct formats, with each format analogous to different components of the cardiac cycle: QRS isointegral map, STT isointegral map, ST0 isopotential map, and ST60 isopotential map. The QRS isointegral map represents the area under the curve for the QRS complex and is therefore a measure of the size of the

Name: William Brady Patient reference: 99999 Recording Time: October 30, 2006 at 15:53 Analysis BeatView
 Age: 29 Gender: Male Race: White
 25 mm/sec 10 mm/mV 0.05–40.0 Hz 60 Hz~

Referring Physician: Brady
 Recording Information
 Hospital: UVA
 Department: Emergency Department

Analysis Information
 Analysed By: AUTO
 Date and Time: October 30, 2006 at 15:56
 Software Revision: 1.7



Heartscape Technologies: PRIME ECG ® Version 1.7:

Figure 14-8 The 80-lead ECG.

QRS complex. The STT isointegral map is the area under the curve of the ST segment and the T-wave; this isointegral thus depicts the ST segment and T wave. The ST0 and ST60 isopotential maps measure voltage at a single time point on the ST segment. The ST0 map measures the voltage at the J point, or juncture, of the QRS complex with the ST segment. The ST60 isopotential map measures the voltage at a single point on the ST segment, 60 msec after the J point.

In the torso maps, colorimetric imaging is used (see Fig. 14-9). Positive structures (i.e., those electrocardiographic waveforms located above the baseline) are indicated by red—either a QRS complex with R-wave, ST segment with elevation, or a prominent, upright T-wave. Electrocardiographic structures found below the baseline would be blue in color—a QRS complex with either prominent Q- or S-waves, ST segment depression, or inverted T-wave. The color green notes a QRS complex that is isoelectric (i.e., no net positive or negative deflection) as well as ST segments and T-waves that are normal.

The body map ECG should not replace the typical 12-lead ECG in the chest pain patient; in fact, the body map ECG should be used only as a second-tier evaluation tool in the consideration of the patient with intermediate to high clinical suspicion for ACS and an unrevealing initial 12-lead ECG. The primary clinical indication for body map ECG performance is the chest pain patient with an intermediate to high clinical suspicion for ACS and a nondiagnostic 12-lead ECG. In this instance, the clinician is in search of an ST segment elevation AMI in electrocardiographically silent areas—namely, the far inferior and lateral walls, the posterior wall, and the right ventricle. Secondary indications include (1) the patient with an initially lower suspicion for ACS and a nondiagnostic ECG who later demonstrates a significantly positive serum marker and (2) the patient with an inferior wall ST segment elevation AMI with additional cardiac segment involvement (e.g., inferoposterior ST segment AMI or inferior ST segment elevation AMI with right ventricular infarction).

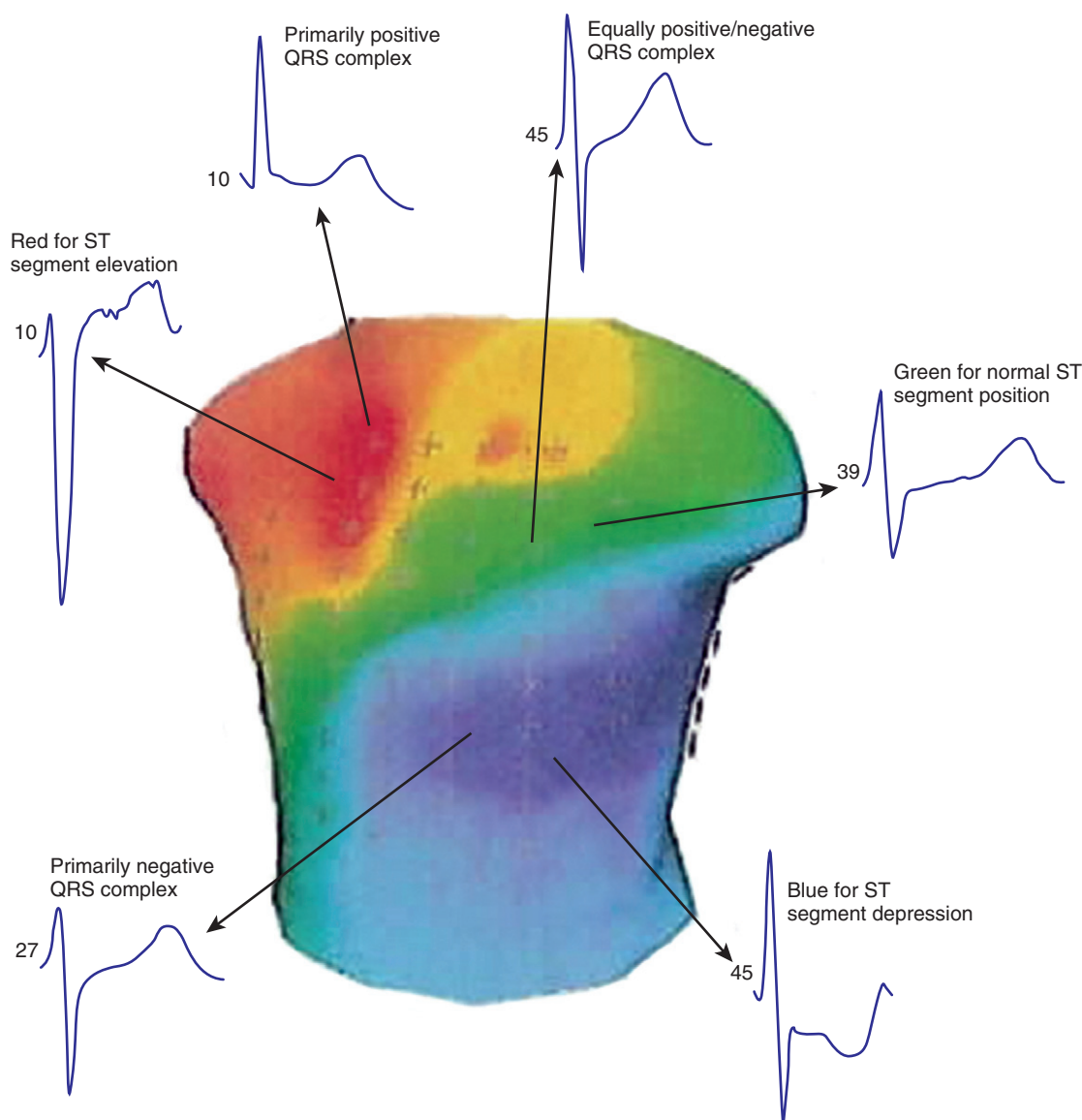


Figure 14-9 Colorimetric interpretation of the torso body map. Green indicates either a normal ST segment (i.e., no elevation or depression) or a QRS complex that has a net negative amplitude (neither positive nor negative), red shows either ST segment elevation or predominantly positive QRS complex, and blue shows either ST segment depression or predominantly negative QRS complex.

ALTERNATIVE LEADS AND TECHNIQUES FOR RHYTHM ASSESSMENT

Electrocardiographic rhythm assessment depends on a clear signal of both atrial and ventricular electrical activity over a period of time. Although continuous 12-lead ECG rhythm monitoring has the advantage of recording cardiac activity over multiple leads (thus maximizing atrial and ventricular monitoring), it is often impractical. Moreover, correct identification of the cardiac rhythm on ECG can be difficult, depending on the clinical setting. Rapid atrial or ventricular rates, especially those above 150 beats/min, often lead to simultaneous or near-simultaneous deflections that can alter the usual waveforms or cause smaller deflections to be buried within larger ones (such as P-waves buried within the QRS complex). In addition, rapid rates result in smaller, narrower waveforms that make visual recognition on the ECG challenging. Finally, assessment of atrial activity in general is more difficult owing to the smaller electrical impulse, and resulting electrocardiographic waveform, generated by the atria.

Lead V₁ is generally considered the best lead for detecting the P-wave, followed by lead II. In a study of 62 measurements in 28 patients, lead V₁ demonstrated the tallest P-wave 53% of the time, followed by lead II (29%), lead I (7%), and lead III (3%).²⁷ A number of alternative techniques have been developed to improve rhythm assessment. These techniques include alterations in the standard 12-lead ECG as well as the addition of nonstandard leads to monitor cardiac, and particularly atrial, rhythm activity.

Alteration in Amplitude and Paper Speed

Most 12-lead electrocardiography machines today allow alteration of both amplitude and paper speed from the basic 10 mm/mV and 25 mm/sec standards, respectively. Increasing the amplitude, most commonly to double the standard or 20 mm/mV, can increase the prominence of smaller deflections, such as the P-wave, and improve recognition of the atrial rhythm (Fig. 14-10). In addition, clinicians have also utilized photocopy enlargements of the standard ECG to visually enhance smaller deflections.²⁸

Increasing the paper speed, again most commonly to double the standard, or 50 mm/sec, has the effect of artificially slowing the rhythm. This technique is most advantageous when assessing patients with marked atrial or ventricular tachycardia. Increasing the paper speed exaggerates any existing irregularity (such as in atrial fibrillation) and can improve recognition of smaller deflections, such as P-waves, in the presence of a significant tachycardia. Faster paper speeds also make it possible to measure short electrocardiographic intervals (such as P-R or R-R) more accurately (Fig. 14-11).

Accardi and coworkers²⁹ found that overall diagnostic accuracy improved when clinicians were provided ECGs recorded at the faster 50-mm/sec paper speed, as opposed to a standard 12-lead ECG, in patients with narrow-complex tachycardias. Moreover, they reported this improved rhythm assessment likely would have resulted in fewer treatment errors.

Alternative Leads

Rhythm assessment often requires electrocardiographic monitoring over continuous periods of time, making the standard 12-lead ECG (requiring 10 electrodes), and even unipolar precordial V₁ monitoring (requiring 5 electrodes), not feasible. A number of alternative lead systems requiring fewer electrodes have been described. Many of these systems utilize the limb bipolar leads (RA, LA, LL) in alternative positions over the chest. Leads I, II, or III are then recorded depending on the positions of the positive and negative electrodes.

Lewis Leads

In 1910, Thomas Lewis³⁰ first described alternative positions for the RA and LL leads to enhance detection of atrial fibrillation. The RA lead was placed over the right second costochondral junction, while the LL lead was placed in the right fourth intercostal space 1 inch to the right of the sternum—leaving the LA and RL leads in their usual positions. Lewis³⁰ reported enhancement of atrial activity when the RA served as the negative electrode and LL as the positive electrode (lead II) with this new configuration. Other alternative lead placements to enhance atrial activity detection have been described³¹⁻³³ (Table 14-2 and Fig. 14-12).

TABLE 14-2 Alternative Leads for Rhythm Assessment

Lead I*	RA = negative electrode	LA = positive electrode
Lead II*	RA = negative electrode	LL = positive electrode
Lead III*	LA = negative electrode	LL = positive electrode
Alternative Lead	Negative Electrode Position	Positive Electrode Position
Lewis [†]	R second costochondral junction	R fourth intercostal space, 1 inch right of sternum
Drury	Second R costochondral junction	Seventh R costal cartilage
	Center of sternum	Inferior angle of scapula 2 inches right of spine
Schoenwald	Third intercostal space along R sternal border	L leg
	Third intercostal space along R sternal border	R arm
Lu	1st intercostal space directly above V ₁	Approximately 3 inches directly below V ₄
Vertical sternal ("Barker" leads)	Below suprasternal notch at manubrium	Xiphoid process
MCL ₁	L shoulder (1 cm inferior to L midclavicle)	V ₁ (fourth intercostal space R sternal border)
MCL ₆	L shoulder (1 cm inferior to L midclavicle)	V ₆ (~sixth rib midaxillary line)

*First, set the electrocardiographic machine to record the rhythm strip using this lead. If the recording rhythm strip is lead I, the RA wire becomes the negative electrode that is placed as noted in table, and the LA wire becomes the positive electrode that is placed as noted in the table. If lead II or lead III is the lead that is set to record the rhythm strip, the positive and negative electrodes will vary.

[†]Example: One way to record the Lewis lead: Set the electrocardiographic machine to record lead I, use the RA wire as the negative electrode, and place it in the R second costochondral junction. Use the LA wire as the positive electrode and place it in the right fourth intercostal space, 1 inch right of the sternum. The Lewis lead may also be recorded on lead II and lead III, but the wires that serve as the positive and negative electrodes will vary.

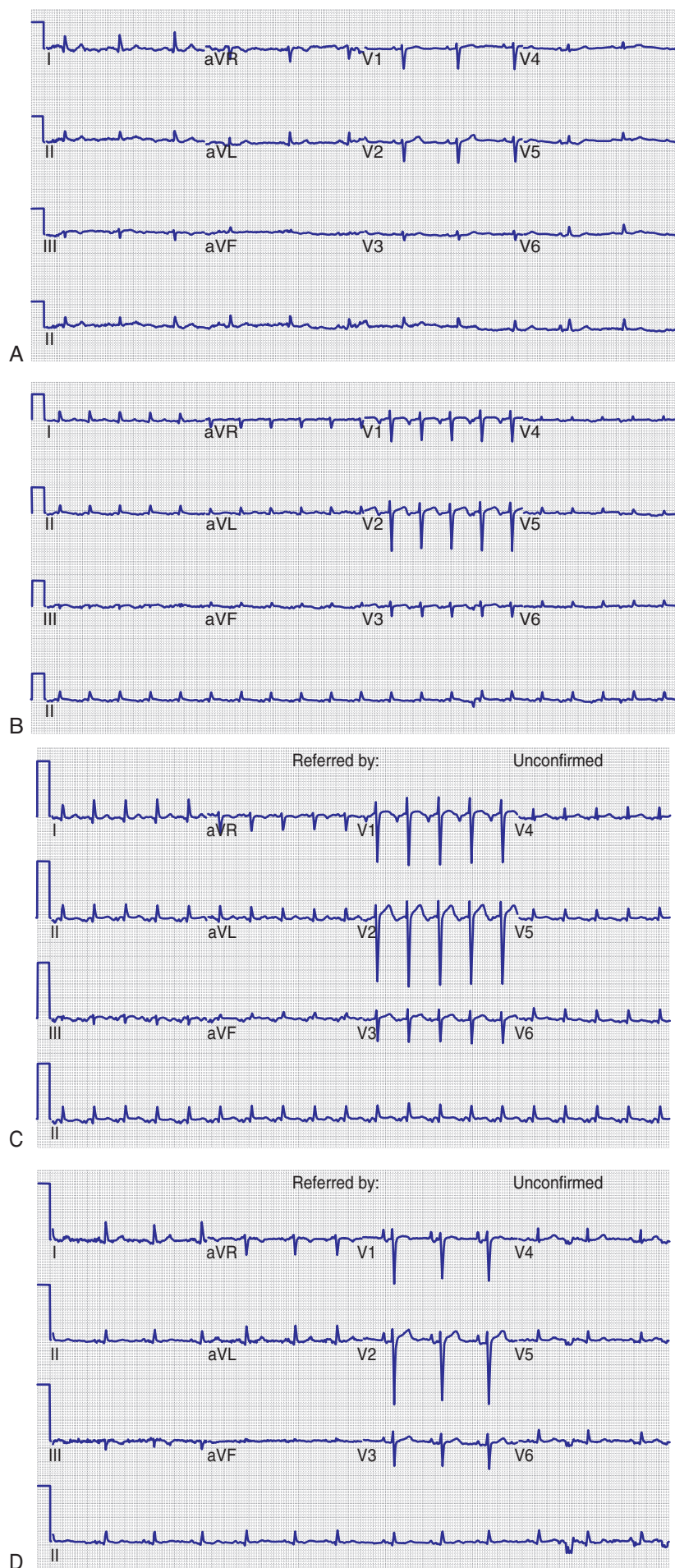


Figure 14-10 A, Baseline ECG of a patient before development of abnormal rhythm (10 mm/mV). Note the P-wave morphologies, especially in leads I, II, and V₁. B, ECG during ectopic atrial tachycardia (10 mm/mV). Note the change in P-wave morphology, especially in lead V₁. C, ECG during ectopic atrial tachycardia (20 mm/mV). The P-waves are now easier to see in all leads. D, ECG after reversion to normal atrial focus (20 mm/mV). Contrast these accentuated P-waves to those in C.



Figure 14-11 A, ECG with tachycardia at normal paper speed (25 mm/sec). Because of the rapid rate, the actual P-waves are difficult to discern, making rhythm determination difficult. The computerized interpretation is sinus tachycardia with first-degree atrioventricular (AV) block. B, ECG with tachycardia at double paper speed (50 mm/sec). With increased paper speed, atrial P-wave activity is accentuated, demonstrating atrial flutter with a 2:1 AV block.

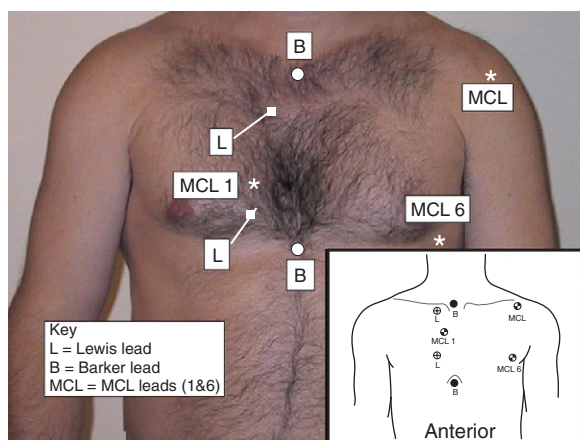


Figure 14-12 Alternative leads. Three of the more commonly used alternative lead strategies for atrial rhythm clarification (Lewis leads, vertical sternal or Barker leads, and modified bipolar chest lead 1 [MCL₁]), and ST/T-wave monitoring (MCL₆).

Vertical Sternal "Barker" Leads

In this alternative lead system, the positive electrode is placed at the xiphoid process and the negative electrode is placed just below the suprasternal notch on the manubrium. Herzog and associates²⁸ reported that vertical sternal leads produce a larger P-wave than other systems, including the Lewis leads. In addition, the vertical sternal leads are placed over bone, which may reduce muscle activity artifact on recordings (see Fig. 14-12).

Limb-Precordium Leads

A sequential pattern of bipolar leads on the chest, termed *limb-precordium leads*, has been proposed in combination with the original Einthoven limb leads. In this system, standard limb leads are placed on the patient. The RA electrode is then repositioned sequentially at the fourth intercostal space just right of the sternum, the fourth intercostal space just left of the sternum (low parasternal), the first intercostal space just left of the sternum (high parasternal), and the first intercostal space just right of the sternum (high parasternal). During this sequential mapping, tracings are recorded for leads I and II until atrial activity is identified. Brenes-Pereira³⁴ reported that this mapping system allowed for the identification of P-waves for a majority of patients when none was detected initially on the standard 12-lead ECG.

Modified Bipolar Chest Leads

Modified bipolar chest leads (MCL) are the most commonly used leads for cardiac rhythm monitoring. The positive electrode is placed on the chest at a precordial position (V) concordant with the MCL desired (e.g., the V₁ position for MCL₁). The negative electrode is placed on the left shoulder. On standard electrocardiographic machines, the LA electrode is placed at V₁, RA at the left shoulder, LL at V₆, and RL at a remote location on the chest to serve as ground. Lead I would then reflect MCL₁ and lead II MCL₆. MCL₁ may be useful in distinguishing atrial activity, MCL₅ and MCL₆ more commonly in ST/T-wave monitoring, and both MCL₁ and MCL₆ in evaluating wide-complex tachycardias³⁵ (see Fig. 14-12).

Esophageal Leads

The esophageal lead (E) was first described by Brown in the 1930s.³⁶ Since that time, both unipolar and bipolar esophageal leads have been developed.³⁷ Because of its posterior location, this lead is often superior at detecting atrial deflections and recording the activity of the posterior surface of the left ventricle. The electrode, which is connected to the ECG by thin wires, is either swallowed or passed through the nares into the esophagus. Once in the esophagus, the location of the electrode is determined either by fluoroscopy or by making a series of low-to-high esophageal recordings. The position of the electrode in the esophagus is adjusted by slowly pulling the electrode wire out the nares or mouth. For the normal adult, leads E_{15–25} (electrode is located in the esophagus 15–25 cm from the nares) generally records atrial activity; E_{25–35}, activity of the AV groove; and E_{40–50}, activity of the left ventricular posterior surface. The E lead should be recorded through lead channel I, simultaneously with the lead channel II and the other surface channels.

Central Venous Catheter Intracardiac Leads

In patients in whom a central venous catheter was placed for vascular access (or for other reasons, such as cardiac pacing, hemodialysis, or Swan-Ganz monitoring), that catheter, when filled with saline, was used as a modified intracardiac electrode for recording of atrial activity. Once filled with saline, a needle was then left in a side access port of the catheter and attached via an alligator clip to lead V₁. Using this method, the distal port of the saline-filled central venous catheter demonstrated significantly larger P-waves than the standard 12-lead ECG and the Lewis lead.²⁷

ELECTRODE MISPLACEMENT AND MISCONNECTION

Limb Electrode Reversals

Whereas the limb electrodes are not often misplaced, the cables that link them to the ECG are at times improperly connected. This can result in “electrocardiographic changes” that are, in actuality, artifacts. A multitude of possibilities for misconnection of the limb electrodes exists; some of the most probable are summarized here. It is helpful to categorize these possibilities into those that are easily recognizable without comparison to an old ECG versus those that are not.

Recognizable without Old ECG

The most common of all misconnections is LA and RA electrode reversal (Fig. 14–13). The hallmark is a negative P-wave and primarily negative QRS complex in lead I, creating a right or extreme axis deviation (depending upon the principal vector of the QRS complex in lead aVF). Dextrocardia should also be considered with this presentation; the pattern of precordial lead transition will differentiate between dextrocardia and arm electrode reversal, however, with dextrocardia featuring progressive diminution in QRS amplitude as the eye moves from lead V₁ (right-sided) toward lead V₆ (left-sided). Moreover, lead aVR is actually aVL in this circumstance, and thus may feature an upright QRS—which is highly unusual for aVR.³⁸ A further clue to arm electrode reversal is the resultant discrepancy in the major QRS vectors of leads I and V₆. Because the vectors of those two leads is leftward, the QRS complexes are expected to point in similar directions when the ECG is performed properly. These two leads will feature discordant QRS vectors when the arm electrodes are reversed (see Fig. 14–13). Transposition of the RA and LL cables is also easily recognized; all leads are upside down compared with the usual patterns, with the exception of aVL, which is unchanged.

Anytime the RL electrode is transposed with another extremity lead, one of the limb leads will appear as virtually a straight line, and thus is easily recognized if this finding is not incorrectly ascribed to poor electrode contact or function. An exception to this rule is if the leg electrodes are reversed (RL ↔ LL), in which case the ECG is virtually identical to one with correct placement of the electrodes. Reversal of the leg electrodes is largely insignificant in that the potentials at the left and right legs are essentially the same.³⁹

Recognizable with Old ECG

One limb electrode reversal that is not readily recognizable without comparison to a prior tracing is transposition of the LA and LL electrodes. This causes reversal of lead I with II on the tracing, as well as aVL with aVF. In effect, two inferior leads (II and aVF) have become the lateral leads (I and aVL), and vice versa—thus making this misconnection difficult to detect, at times, without a baseline ECG for comparison. Furthermore, lead III will be upside down (although a negative QRS complex in III is not unusual), and aVR will be

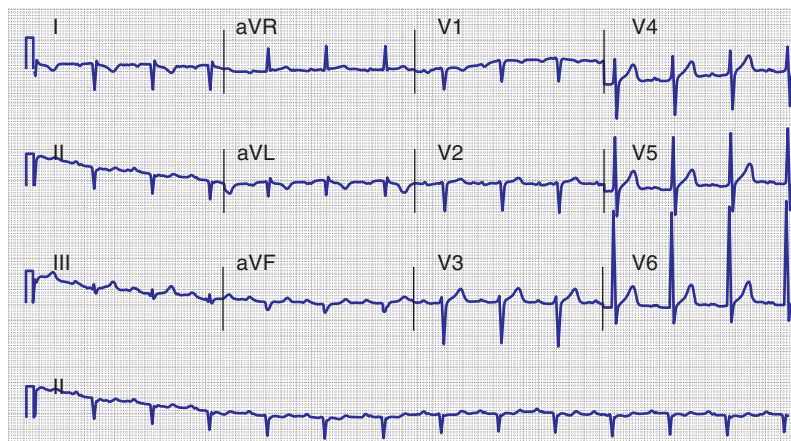


Figure 14–13 Arm lead reversal (LA ↔ RA). The most common of limb lead reversals, the clues lie in leads I and aVR. Lead I features a negative P-wave as well as a principally negative QRS complex and T-wave. This could suggest dextrocardia, but the precordial leads demonstrate normal transition, which is not consistent with dextrocardia. Note also the unusual appearance of aVR in this tracing.

unchanged (Fig. 14-14).³⁹ Suspect LA/LL reversal when comparing two ECGs that feature changes that do not make clinical sense; if the P-QRS-T wave morphologies in lead III in the two tracings are mirror opposites, repeat the ECG with close attention to correct electrode connection.

Clues to limb electrode reversal are summarized in Table 14-3.

Precordial Electrode Misplacement and Misconnection

Unlike the limb electrodes, the precordial electrodes are more prone to misplacement, especially when variations in body habitus (e.g., obesity, breast tissue, pectus excavatum, chronic lung disease) make proper electrode placement more difficult.



Figure 14-14 A, Limb lead reversal (LA ↔ LL). A patient with a history consistent with acute coronary syndrome was brought to the emergency department after this ECG was recorded in a clinic. Leads I and aVL suggest an acute high lateral infarct but, surprisingly, there are no corresponding changes in leads V₅ and V₆. The deep T-wave inversions in III and aVF were at first thought to be inferior ischemia or reciprocal changes (see also B). B, Correction of lead reversal (LA ↔ LL). After the leads were reconnected, this tracing reveals an acute inferior wall myocardial infarction (MI), as well as deep T-wave inversion in aVL—a harbinger of acute inferior MI. Comparing this tracing with that in A, note the following: lead I ↔ lead II; lead aVL ↔ aVF, and lead III is inverted. Thus, inferior changes become lateral, and lateral become inferior.

TABLE 14-3 Clues to Improper Limb Lead Connections		
Reversed Leads	Old ECG Necessary for Detection?	Key Findings
LA RA	No	PQRST upside down in lead I Precordial leads normal (not dextrocardia)
LA LL	Yes	III is upside down I ↔ II; aVL ↔ aVF; aVR no change
LA RL	No	III is straight line
RA LL	No	PQRST upside down in all leads except aVL
RA RL	No	II is straight line
LL RL	Cannot detect change	Looks like normal lead placement
LA LL } RA RL }	No	I is straight line aVL, aVR are same polarity and amplitude and II is upside down III

LA, left arm; LL, left leg; RA, right arm; RL, right leg.
From Surawicz B, Knilans TK: Chou's Electrocardiography in Clinical Practice, 5th ed. Philadelphia, WB Saunders, 2001.

This may cause some variability in the amplitude and morphology of the complexes in the precordial leads. However, these changes are not usually grossly abnormal, and therefore can be difficult to detect. Variation often becomes evident when comparing the current tracing with an old ECG.³⁹ In such cases, it is useful to go to the bedside and examine where the electrodes were positioned relative to the recommended placement (see “[Electrode Placement](#)” earlier in this chapter). One cannot ensure, however, that the baseline ECG was done with proper electrode placement. When comparing the precordial leads on the current ECG with a baseline tracing, ST segment and T-wave changes should be viewed in the context of the relative morphologies of the associated QRS complexes. If there is a marked difference between the two tracings in the amplitude and polarity of the QRS complex in a given precordial lead, the corresponding ST-T wave changes may be due to variability in electrode placement—although cardiac ischemia cannot be completely excluded as the cause.

Misconnection of the precordial cables is usually easy to detect. The expected progression of P-, QRS, and T-wave morphologies across the precordium will be disrupted ([Fig. 14-15](#)). An abrupt change in wave morphology evolution—followed by a seeming return to normalcy in the next lead—is a good clue to precordial electrode misconnection.³⁹

ARTIFACT

Electrocardiographic artifact is commonly encountered, yet not always easy to recognize. It can be attributed to either

physiologic (internal) or nonphysiologic (external) sources; the former includes muscle activity, patient motion, and poor electrode contact with the skin. Tremors, hiccups, and shivering may produce frequent, narrow spikes on the tracing, simulating atrial and ventricular dysrhythmias^{38,40} ([Fig. 14-16](#)). A wandering baseline featuring wide undulations, as well as other “noise” on the ECG, can often be traced to patient movement and high skin impedance, leading to inadequate electrode contact to the skin. Minimizing skin impedance and artifact may be achieved by (1) avoiding electrode placement over bony prominences, major muscles, or pulsating arteries, (2) clipping rather than shaving thick hair at electrode sites, and (3) cleaning and, most importantly, drying the skin surface before reapplying the electrode if the tracing features substantial artifact.^{38,41} Nonphysiologic artifact is most often due to 60-Hz electrical interference, which is ascribable to various other sources of alternating current near the patient. This will manifest as a wide, indistinct isoelectric baseline. Other sources of nonphysiologic artifact include loose connections, broken monitor cables, and mechanical issues with the machine (e.g., broken stylus, uneven paper transport). The 60-Hz artifact due to electrical current interference can be minimized by shutting off nonessential sources of current in the vicinity as well as straightening the lead wires so that they are parallel to the patient’s body in the long axis.^{38,40,42}

Differentiation of artifact from true electrocardiographic abnormality is intuitively important; moreover, clinical consequences have been reported that are directly attributable to confusion of artifact with disease. Unnecessary treatment and procedures—including cardiac catheterization, electrophysio-

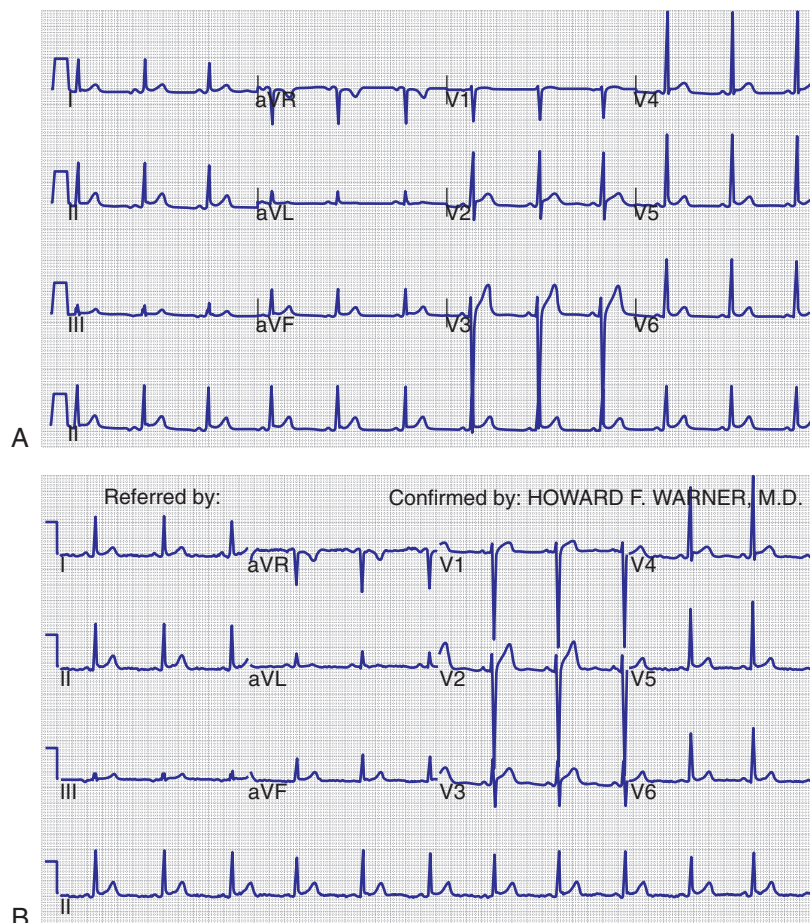


Figure 14-15 A and B, Precordial lead reversal ($V_2 \leftrightarrow V_3$). Note that the usual precordial progression of R-wave growth in leads V_2 and V_3 is disrupted in the tracing displayed in A. B shows a return to a normal V_3 transition zone.

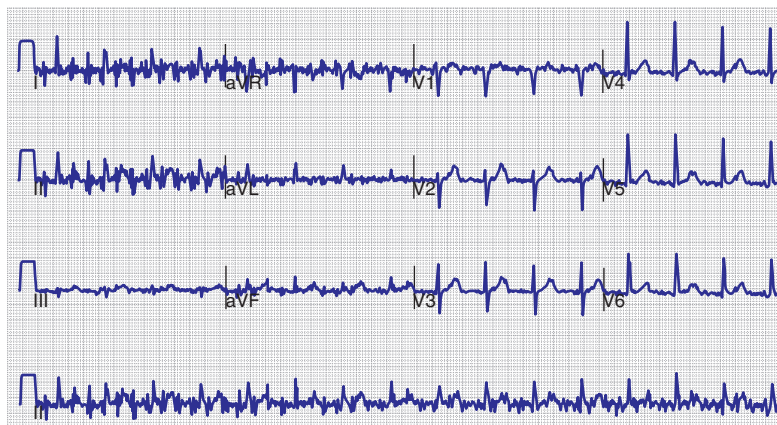


Figure 14–16 Artifact due to physiologic cause. The patient's monitor was alarming owing to a perceived heart rate of greater than 200 beats/min, and the computerized alert system called this ventricular tachycardia. The patient, who has Parkinson's disease, was without complaint. The ECG demonstrates a marked artifact, giving the appearance of atrial flutter in lead V₁.

logic testing, and even implantation of a pacemaker and an automatic defibrillator—have been reported.⁴³ Characteristics that may aid in differentiating artifact from dysrhythmia include absence of hemodynamic instability during the event (or even absence of any symptoms); normal QRS complexes occurring during the dysrhythmia; instability of the baseline on the tracing during and immediately after the “dysrhyth-

mic” event; association with body movement; and observance of “notches” amidst the complexes of the pseudodysrhythmia which “march out” with the normal QRS complexes that precede and follow the disturbance.^{44,45}



REFERENCES CAN BE FOUND ON EXPERT CONSULT