

How many ECG leads do we need?

Elin Trägårdh, MD, PhD candidate*,
Henrik Engblom, MD, PhD candidate, Olle Pahlm, MD, PhD

Department of Clinical Physiology, Lund University Hospital, SE-221 85 Lund, Sweden

The number of leads used and the electrode placement in a standard ECG have remained the same for over half a century although the technology behind the method has developed greatly (Fig. 1). It is well known that the sensitivities of current standard 12-lead ECG criteria for detecting many cardiac diseases—such as acute myocardial infarction (MI) and ventricular hypertrophy—are poor. To increase the diagnostic ability of the ECG, many alternative lead systems have been proposed, but none has yet received general acceptance in the cardiology community.

The issue of the optimal number of leads is of great interest to researchers, as becomes apparent when researching the literature on the subject. Some investigators argue that a small number of leads will suffice; others maintain that the greater the number of leads, the better. This article aims to answer the question of how many ECG leads actually are necessary by presenting some of the most widely studied lead sets. Also discussed are researchers' efforts to enhance the sensitivity and specificity of the ECG in detecting acute MI and the capability of the ECG to diagnose other cardiac conditions.

The development of the standard 12-lead ECG

The first surface ECG recorded on humans was performed in 1887 by Waller [1], but the leads that were first used for diagnostic electrocardiography were three-limb leads introduced by Einthoven [2]. In the earliest ECG recording systems, the patient's arms were inserted into jars of conducting solution, which were connected to a sensitive galvanometer. This method made it possible to measure the potential differences between the

two arms (lead I), the left leg and the right arm (lead II), and the left leg and the left arm (lead III).

In the early 1930s, Wilson [3,4] introduced a “central terminal,” which he hypothesized would allow measurements of the potential variation at a single point, giving rise to the incorrect term “unipolar leads.” Indeed, all leads measure the potential difference between two poles. The original Einthoven leads were “two-electrode leads,” whereas other standard leads used a single electrode as one pole and averaged inputs from multiple electrodes as the second pole. When inputs from all three limb electrodes were averaged, the resulting “central terminal” served as the second pole, thus creating a “V” lead. The lead system defined six additional chest leads to be recorded, labeled V1, V2, V3, V4, V5, and V6.

In the early years of ECG recordings, there was no standardization of placement of the precordial leads, making it difficult to compare studies. In 1938, the first standardization was made by the Cardiac Society of Great Britain and Ireland in conjunction with the American Heart Association [5]. Later that same year, the electrode positions of leads V1 to V6 as known today were described [6,7]. The standardized placement of the precordial leads was a committee decision, made with the specific intent of standardizing research.

The Wilson central terminal also enabled three other limb leads: VR, VL, and VF. These leads, however, were of low voltage in general and were replaced by augmented (aV) leads by removing input from the electrode that provided the first pole from the averaged second pole. Augmented limb leads, developed in 1942, measure the potential differences between the left arm and the average of the potentials at the right arm and left leg (lead aVL). In a similar fashion, it was possible to design two other augmented limb

* Corresponding author.

E-mail address: elin.tragardh@med.lu.se (E. Trägårdh).

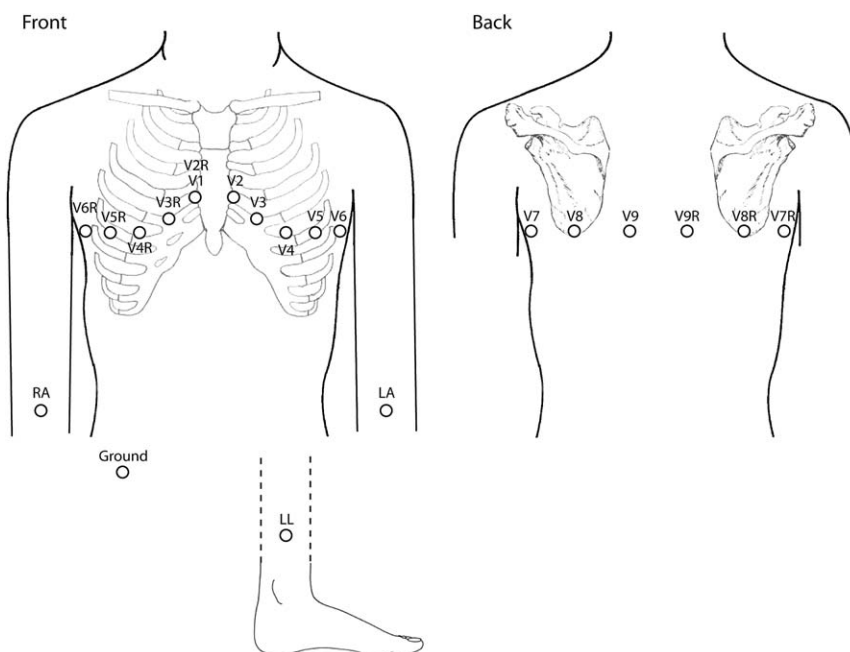


Fig. 1. The placement of precordial and limb electrodes. In the frontal view (left) the electrode placement for the standard 12-lead ECG is shown: V1 to V6. The right-sided electrodes are also shown, which are mirror images of leads V2 to V6. On the back view (right) the posterior electrodes V7 to V9 and V7R to V9R are shown. LA, left arm; LL, left leg; RA, right arm.

leads, aVR and aVF [8], creating the standard 12-lead ECG used today.

In some situations it is not feasible to use the standard electrode placement for limb leads because of noise from skeletal muscle (eg, during percutaneous transluminal coronary angioplasty and during stress tests). In 1966, Mason and Likar [9] introduced a system in which limb electrodes are instead placed on the torso (Fig. 2). In this system, the arm electrodes are placed in the left or right infraclavicular fossa, medial to the border of the deltoid muscle and 2 cm below the lower border of the clavicle. The left leg electrode is placed in the anterior axillary line, halfway between the costal margin and the crest of the ilium. Studies have shown, however, that the electrical axis may change and that ECG findings indicating inferior or posterior infarcts are lost in 69% and 31% of patients, respectively, compared with findings in ECGs recorded with standard electrode placement [10,11]. Hence, when interpreting an ECG, it is important to know if it has been recorded using Mason-Likar electrode placement.

Additional precordial leads

It is well known that the sensitivity of current 12-lead ECG criteria is poor for detecting acute MI

[12,13]. There are three principal reasons. First, the infarct may be too small to affect the cardiac electrophysiology reflected by an ECG. Second, the location of the infarction might not be “observed” by a 12-lead ECG because of its limited coverage of the precordium. It also is possible that the precordial electrodes in a standard 12-lead ECG are not optimally placed to detect acute MI and ischemia. For example, Saetre and colleagues [14] found that leads derived from electrodes placed in the left axilla and on the back could differentiate ECG changes that occurred during percutaneous coronary intervention of the first diagonal and left circumflex (LCX) arteries, respectively.

Third, the location of ischemia in acute infarction might be observed by the 12-lead ECG as ST depression when, in fact, it should be considered “ST elevation equivalent.” This consideration of the inverse of a standard lead as the equivalent of an additional lead (eg, lead aVR and lead –aVR) may negate the need to create additional leads by placement of additional electrodes [15].

In addition to precordial leads V1 to V6, other precordial torso leads can be used as well. Posterior leads V7 (left posterior axillary line), V8 (left midscapular line), and V9 (left border of the spine) are all located in the same horizontal plane as lead

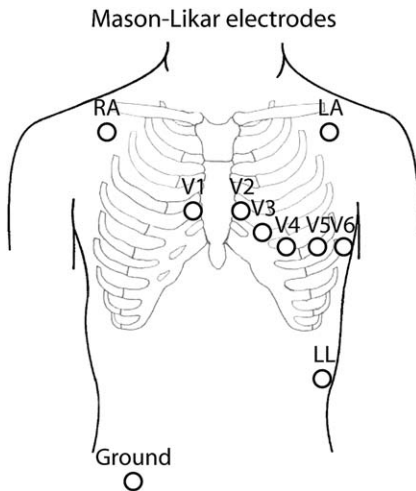


Fig. 2. Mason-Likar electrode placement. In comparison with the electrode placement for the standard 12-lead ECG, the limb leads are moved proximally when recording the 12-lead ECG according to the Mason-Likar system. The upper limb leads are placed in the infraclavicular fossa medial to the border of the deltoid muscle and 2 cm below the lower border of the clavicle. The lower limb lead is moved to the anterior axillary line, halfway between the costal margin and the crest of the ilium. LA, left arm; LL, left leg; RA, right arm.

V6. Leads V3R to V9R are located on the right side of the chest in the same location as corresponding to the location of the left-sided leads V3 to V9. V2R is therefore the same as V1 (Fig. 1).

Posterior leads

Posterior MI occurs when either the LCX or a branch of the right coronary artery (RCA) supplying blood to the posterior wall of the left ventricle is occluded. Because there are no posterior electrodes in a standard 12-lead ECG, the diagnosis of posterior MI depends on ST depression in anterior leads [16]. Currently, patients who do not have ST-segment elevation in a standard ECG are not recommended for thrombolytic therapy [17]. Moreover, there are reports of ST elevation in posterior leads in the absence of changes in anterior leads [18]. It is important to diagnose posterior MI because it often is associated with complications such as mitral valve regurgitation [19] and poor patient prognosis [20,21]. Accordingly, there has been much interest in the study of posterior chest leads in the past few years.

In one such study, ECG patterns in posterior chest leads (V7–V9) were studied in 225 normal male subjects about to undergo military training

[22]. The investigators found that none of the subjects had ST elevation at the J point exceeding 0.5 mm. The prevalence of 0.5- to 1.0-mm ST elevation at 80 milliseconds after the J point in lead V7 was 8.9%, 5.8% and 3.1% in leads V7, V8, and V9, respectively. In only two subjects did the ST elevation reach 1 mm in amplitude. The results indicate that ST elevation in leads V7 to V9 at the J point or of more than 1 mm 80 milliseconds after the J point can be considered abnormal.

In acute MI, it is important to open the occluded artery as soon as possible after the onset of symptoms. It is desirable to identify the culprit artery by surface ECG to facilitate coronary intervention. A prospective ECG analysis during angioplasty for single-vessel disease involving LCX or RCA found that the most common ECG change during RCA occlusion was ST-segment elevation in leads II, aVF, and III (95%), and that the most common change during LCX occlusion was posterior ST elevation in leads V7 to V9 (68%) [23]. Therefore, the use of posterior leads may help in distinguishing between occlusion of the RCA and occlusion of the LCX.

Many studies have demonstrated that posterior leads are useful for identifying acute posterior MI not detected by the standard ECG [19,21,24–26]. Zalenski and colleagues [26] found that the addition of V4R, V8, and V9 in patients admitted to a cardiac-monitored unit with a provisional diagnosis of MI or unstable angina increased the sensitivity of ST-segment elevation detection from 47.1% to 58.8%, with no loss of specificity. Biochemical markers were used to diagnose MI, and percutaneous coronary intervention was not performed in the study.

In another study, however, Zalenski and colleagues [27] found that the accuracy of detecting ST-segment elevation in acute MI was improved only modestly by the addition of leads V7 to V9. The sensitivity increased from 57.7% to 59.7%, whereas the specificity decreased from 91% to 89.4%.

In a study of low-risk patients presenting with chest pain suggestive of acute coronary syndromes, posterior leads were not found to increase the detection rate of ischemia [28]. Low-risk patients were identified by a normal 12-lead ECG, absence of arrhythmias or hemodynamic instability, and one negative serum cardiac troponin I assay. Different studies have used different levels of ST elevation in posterior leads as diagnostic of acute MI. Some authors have used a criterion of 1 mm, whereas studies with an angiography gold

standard have considered 0.5 mm of ST elevation to be diagnostic [29].

Right-sided leads

Right ventricular infarction rarely occurs in isolation [30] but occur in more than 30% of cases of inferior left ventricular MI [31]. Patients who have right ventricular infarct involvement have a worse prognosis [32,33], a significantly higher incidence of depressed right ventricular function [34], and a high risk of developing high-degree atrioventricular nodal block compared to infarcts with no right ventricular involvement [35]. Usually, there are only inconclusive signs of right ventricular infarction in the standard 12-lead ECG. Chou and colleagues [36] concluded that ST elevation of 1 mm or more in lead V1, in the absence of other explanations for the ST elevation, should raise suspicion of right ventricular involvement. Other studies [37–39], however, do not support this criterion because of its low sensitivity. One method that can be used to observe the right ventricle better is to apply right-sided leads. Andersen and colleagues [40] described ST deviations in the right chest leads V3R to V7R during transient balloon occlusion of the coronary arteries. They found that ST elevation always evolved in leads V5R and V6R when the RCA was occluded and was 100% discriminative for RCA occlusion compared with occlusion of either of the two left coronary arteries. ST elevation within normal range or ST depression could be seen in leads V3R and V4R during RCA occlusion.

Morgera and colleagues [41] studied 82 normal subjects and found that an rS pattern was always present in V3R and was present in V4R in 91% of subjects. qR or qS patterns were seen in V6R in 26% of the subjects, in V5R in 10% of subjects, and in V4R in 2% of subjects. In a study by Andersen and colleagues [42], 109 normal subjects were studied. An rS configuration was found in V3R in 98% and in V4R in 91%. A qS configuration was found most frequently in V6R (16%). They also investigated the ST deviation 40 and 80 milliseconds after the J point. Measurements of the ST segment made 80 milliseconds after the last QRS deflection showed significantly more deviation from the isoelectric line than measurements made 40 milliseconds after the last QRS deflection. They concluded that ST-segment deviation should be measured 40 milliseconds after the last QRS deflection and should be at least 0.6 mm to be considered abnormal.

Many studies have evaluated right-sided precordial leads in acute MI and have found a high but variable sensitivity for detecting right ventricular MI [27,37–39,41,43]. Croft and colleagues [37] studied 33 patients who had enzymatically confirmed MI, using myocardial scintigraphy as the criterion gold standard. They found that ST elevation of 1 mm or greater in one or more of leads V4R to V6R is both highly sensitive (90%) and specific (91%) in identifying acute right ventricular infarction. Braat and colleagues [38] reported similar results, with a sensitivity of 93% and a specificity of 95% in lead V4R, which was found to be the best right-sided lead for detecting right ventricular MI. Later, they also performed a study using angiography as the criterion gold standard [43]. Forty-two patients who had inferior MI were included, 17 of whom had right ventricular MI. The sensitivity for detecting right ventricular MI was 100% for lead V4R, and the specificity was 87%. Lopez-Sendon and colleagues [39] found 100% sensitivity and 68% specificity in detecting right ventricular MI in lead V4R.

Another study compared the standard 12-lead ECG with 12-lead ECG plus the addition of V4R to V6R in 533 patients who had chest pain [27]. They found that the sensitivity increased from 57.7% to 64.4% with the addition of V4R to V6R. The specificity, however, decreased from 91% to 85.6%. Most of the studies used 1 mm as the cut-off value [27,37,38,41,43], but others have used 0.5 mm [39,41]. An autopsy study by Morgera and colleagues [41] compared sensitivity and specificity when using a 1-mm or 0.5-mm cut-off in lead V4R. With 0.5 mm as the cut-off, the sensitivity was 76%, and the specificity was 86%. With 1 mm as the cut-off, the sensitivity was 57%, and the specificity was 100%.

The use of right-sided precordial leads has also been investigated during exercise tests, with divergent results. Some studies have shown that the addition of right-sided leads does not increase the sensitivity of the ECG for the detection of myocardial ischemia [44,45], but another study showed that the sensitivity improved greatly [46]. Some studies indicate that the addition of right-sided chest leads could be of use in the early diagnosis of acute pulmonary embolism [47,48].

Body surface potential mapping

The standard 12-lead ECG is derived from only 10 electrodes. Hence, it offers a limited

coverage of the body surface potential distribution caused by the depolarization and repolarization of the myocardium. Therefore, local electrical events in the myocardium may not be reflected in the standard 12-lead ECG. Body surface potential mapping (BSPM) is a method that provides better coverage of the body surface potential distribution by performing recordings at multiple sites (24–240) on the body surface. This approach is not novel; in fact, the first example of a potential map was published in 1889 by Waller [49], who performed 10 to 20 ECG recordings at various points on the surface of the human body. Since then, normal BSPM patterns have been established [50,51], and abnormal patterns of BSPM have been explored. In addition to this empiric use of BSPM, aimed at obtaining descriptive information of cardiac electrical behavior, BSPM has evolved from the so-called “classical forward” and “inverse” problems of electrocardiography. The former focuses on how the electrical activity from electrical sources present in the heart is propagated to the epicardium (near-field problem) or to the body surface (far-field problem) [52]. The latter focuses on how the electrical activity recorded at the body surface can be used to derive details about the electrical activity in the heart (ie, epicardial potentials) [53]. Both problems require information of high spatial resolution from the body surface, which can be obtained through BSPM. The theoretical aspects of BSPM are not discussed further in this article. Instead, the more clinical aspects of BSPM are discussed.

Clinical implications

Several studies involving BSPM in patients with MI have been published [54–74]. A study by Kornreich and colleagues [55] included 177 patients who had MI and 184 normal subjects. Criteria based on six features from three locations (mostly ST-T measurements) derived from 120-lead BSPM data yielded a specificity of 95% and sensitivity of 95% for diagnosing MI. The three locations were the right subclavian area, the left posterior axillary region, and the left leg. The 12-lead ECG had the same specificity but a sensitivity of 88% for the same number of features. The leads with the greatest discriminating power were found outside the 12-lead ECG. Furthermore, it has been shown that the sensitivity for right ventricular MI increased from 42% for right ventricular leads to 58% for BSPM [57]. In the

same study, the sensitivity increased from 1% for posterior leads to 27% for BSPM.

The 12-lead ECG has a sensitivity of about 50% for detection of acute MI. Menown and colleagues [56] studied 635 patients who had chest pain, of whom 325 had MI (according to the World Health Organization criteria for acute MI), and 125 controls. Subjects were randomly assigned to either a learning or a test set. The 64-lead anterior BSPM data showed an overall correct acute MI classification of 74%.

The standard 12-lead ECG is known to have low sensitivity for detection of acute MI in the presence of left bundle branch block. Sgarbossa and colleagues [75] showed a sensitivity of 33%, and Hands and colleagues [76] showed a sensitivity of 17% for detecting acute MI in the presence of left bundle branch block. Maynard and colleagues [58], however, have shown that the sensitivity can be increased to 67% using BSPM.

BSPM also has been shown useful in evaluating the clinical efficacy of reperfusion therapy [77,78]. In a study of 67 patients who had acute MI undergoing coronary angiography 90 minutes after fibrinolytic therapy, Menown and colleagues [77] showed that BSPM had a sensitivity of 97% and a specificity of 100% for detecting patients who had established reperfusion (Thrombolysis in Myocardial Infarction [TIMI] grade 2–3 flow). In comparison, all ST-resolution patterns on the standard 12-lead ECG showed a sensitivity of 59% and a specificity of 50%.

The 12-lead ECG is used frequently for assessing stress-induced ischemia in coronary artery disease. Using BSPM, Manninen and colleagues [79] showed that regional stress-induced ischemia can be identified at sites on the body surface not covered by the standard 12-lead ECG. Montague and colleagues [80] demonstrated that ischemic repolarization changes were detectable and quantifiable by BSPM at low levels of cardiac stress in patients who had one-vessel disease when the usual ECG criteria of myocardial ischemia frequently were absent. Furthermore, Boudik and colleagues [81] showed that BSPM during dipyridamole stress can be used to differentiate between normal subjects, patients who have syndrome X, and patients who have coronary artery disease. The sensitivity and specificity for detecting syndrome X were 71% and 78%, respectively.

BSPM also has been shown to increase the diagnostic accuracy of left ventricular hypertrophy criteria [82] as well as the ability to estimate left ventricular mass from an ECG [83,84] in

comparison to a standard 12-lead ECG. In addition, BSPM has been shown to be useful in identifying accessory pathways in patients who have Wolff-Parkinson-White syndrome [85–91].

Why is body surface potential mapping not clinical routine?

It is clear that BSPM provides more information regarding the cardiac electrical field than the standard 12-lead ECG. Why then is BSPM not more clinically established? First, recording the large number of leads and displaying them appropriately is time consuming, making the procedure unsuitable for clinical settings. This obstacle has, to a large extent, been overcome by the development of electrode strips that are easier to apply and by modern computer technology. Modern BSPM systems can acquire and process maps in less than 30 seconds. Second, the great amount of information received from BSPM has been difficult to interpret and classify correctly; however, a variety of statistical methods have been devised that allow measurements of cardiac patients to be compared with those of healthy subjects of various ages, sex, and body habitus. Interpretation schemes are now available, also. Nonetheless, correct interpretation and classification of maps remain challenging tasks and require a deep understanding of body surface potential distribution. In a recent study of 389 patients presenting to an emergency department with chest pain, Carley and colleagues [92] showed that BSPM offered a relatively small increase in sensitivity (from 40% to 47.1%), and a comparable decrease in specificity (from 93.7% versus 85.6%) for detecting acute MI in comparison to a standard 12-lead ECG. In this study, the BSPM maps were interpreted by emergency physicians trained in BSPM, not by cardiologists with a special interest in BSPM. Additionally, this study was performed in an emergency setting with unselected patients, thus reflecting the clinical reality of a typical emergency department.

Although BSPM offers better coverage of the body surface potential distribution, further studies of the diagnostic capability of BSPM in different clinical settings are needed to establish it as a clinical routine examination.

Reduced lead sets

Although there are clear advantages in using additional leads in certain circumstances,

problems might occur even when using only the 10 electrodes of the standard 12-lead ECG. Multiple electrodes and wires interfere with auscultation, echocardiograms, resuscitation efforts, and chest radiographs. Noise levels created by limb movement detected by multiple leads make interpretation difficult, and the discomfort to patients caused by so many electrodes tends to be high. Additionally, rapid and accurate electrode placement can be difficult in emergency situations. Fewer electrodes placed at more easily accessed locations could facilitate ECG acquisition for both patient and staff.

Frank leads

Today, the standard 12-lead ECG is the most common method for studying the electrophysiology of the heart. Vectorcardiography is another method that was first described in the early twentieth century. A vectorcardiogram traces the sum of the heart's electrical activity in a three-dimensional space throughout the cardiac cycle. Three orthogonal leads, V_x, V_y, and V_z, are used. The most commonly used and best-studied orthogonal lead system is that of Frank [93]. In this lead system, seven electrodes are used (Fig. 3, upper panel). Five of the electrodes are positioned around the heart on the torso (on the left and right side of the thorax, on the sternum, on the back, and on the left side of the chest, at an angle of 45° with respect to the center of the thorax), one electrode is positioned on the back of the neck, and one on the left foot. From the contributing measurements of these leads, the orthogonal leads V_x, V_y, and V_z are derived. Typically, the leads are analyzed in pairs and presented as loops. The amplitudes of the two leads are measured at the same time and plotted in a diagram, with the amplitude of one lead appearing on the *x*-axis and the amplitude of the other lead appearing on the *y*-axis (Fig. 4). The leads are combined pair-wise to a frontal plane (*x* and *y*), a transverse plane (*x* and *z*), and a sagittal plane (*y* and *z*). The shape, direction of rotation, and area of the loop are then analyzed.

Continuous vectorcardiographic registration with Frank leads has been shown to have substantial potential for monitoring patients who have acute MI [94,95] and may help in identifying candidates for emergency coronary angiography [95]. It also has been shown to correlate more closely with enzymatically estimated infarct size in patients who have Q-wave infarction than

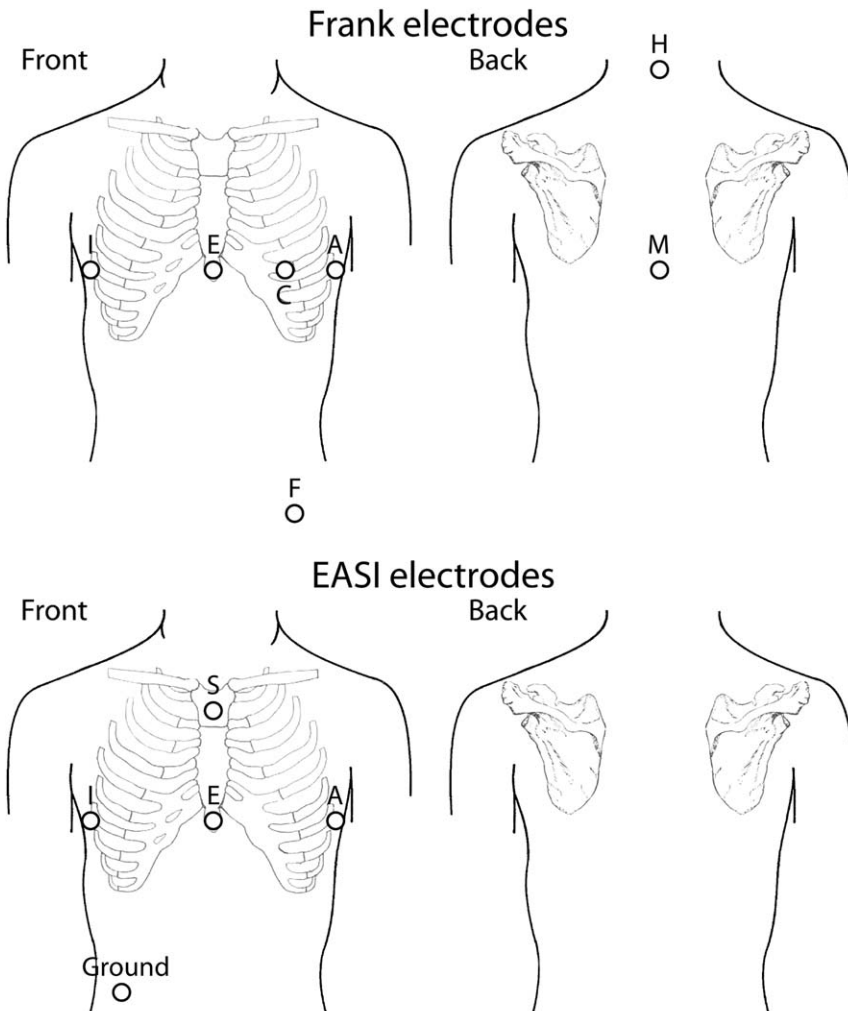


Fig. 3. Comparison between Frank and EASI electrode placement. The E, A, and I electrodes are identical for the two placements. In the EASI setting, however, no electrodes are required on the back.

does QRS scoring of the standard 12-lead ECG [96]. The Common Standards for Quantitative Electrocardiography working party, however, concludes that vectorcardiography has slightly lower specificity and although a slightly higher sensitivity than standard 12-lead ECG [97].

Derived 12-lead ECG

In 1988 Dower [98] introduced a “derived” 12-lead ECG, using five torso electrodes, four recording and one ground. Because three electrodes from the original Frank lead configuration

(E, A, and I) were used, he labeled it the “EASI 12-lead ECG” (Fig. 3, lower panel). In addition, he used a fourth electrode (S). The fifth ground electrode can be placed anywhere. The electrodes are placed on easily identified anatomic points on the torso: the E electrode on the lower sternum, the A and I electrodes on the left and right mid-axillary lines, respectively, at the same transverse level as E, and the S electrode on the sternum manubrium. The electrodes record three ECG leads: A-I (horizontal vector), E-S (vertical vector), and A-S (anterior-posterior vector). Each of the 12 standard ECG leads is derived as a weighted linear

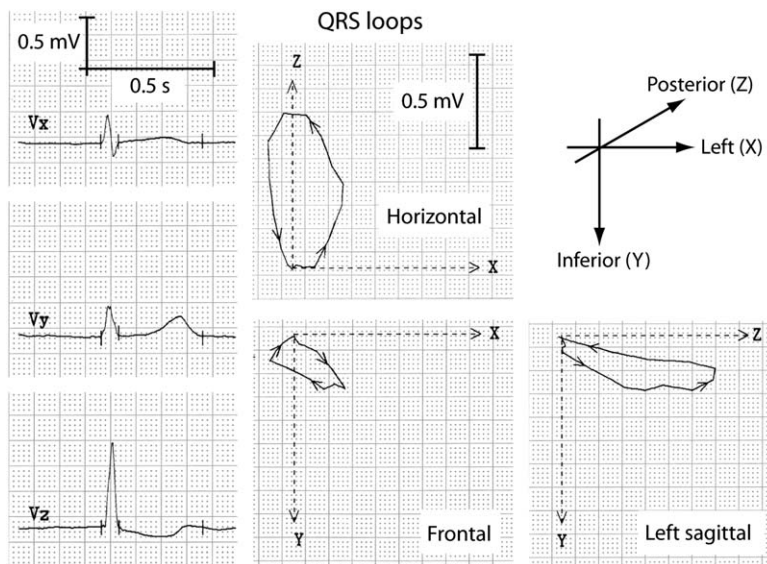


Fig. 4. A vectorcardiogram from a patient who had a large anterior myocardial infarction. To the left the three orthogonal leads Vx, Vy, and Vz are shown. From these leads the QRS loops shown to the right in the frontal, horizontal, and left sagittal planes were derived. As seen in the horizontal and left sagittal planes, the anterior electrical forces are completely absent as a result of the large anterior infarction.

sum of the signals from these three leads using the following formula:

$$L_{\text{derived}} = a(A - I) + b(E - S) + c(A - S),$$

where L represents any ECG lead and a , b , and c represent coefficients determined to optimize the fit between the standard 12-lead ECG and the derived ECG [99].

Basic comparisons between standard 12-lead ECG and EASI-derived 12-lead ECG have concluded that differences between PR, QRS, QT, and QTc intervals are small. Some differences in cardiac axes, especially in T-wave axes, have been found, but EASI still accurately detected right/left bundle branch block and fascicular blocks [100,101].

Accurate diagnosis of both myocardial arrhythmias and ischemia often requires analysis of multiple leads. Many hospital wards, however, rely on only two leads (most often leads II and V1) for ECG monitoring. Continuous monitoring of the EASI-lead ECG has been found to be superior to monitoring with leads II and V1 for diagnosing transient myocardial ischemia in patients who have unstable coronary syndromes [102]. Leads II and V1 were shown to miss 64% of ischemic ST changes detected by EASI; 75%

of the missed events were clinically silent. Moreover, EASI-derived ECG has been found to be diagnostically equivalent to standard 12-lead ECG for the detection of cardiac arrhythmias [100,101].

The diagnostic capabilities of the EASI system have been found to be equivalent to the standard 12-lead ECG for the detection of acute ischemia [101–104], acute MI [105], and prior MI [100,101,103,106]. Another application might be in the ambulance setting. Sejersten and colleagues [107] showed that the EASI lead system can provide an alternative to the standard 12-lead ECG to facilitate data acquisition and possibly save time in emergency situations. So far, few studies have been performed in children, where it also is important to simplify ECG recordings. A basic study by Pahlm and colleagues [108] concluded that EASI leads in children have the same high levels of “goodness-of-fit” to replicate conventional 12-lead ECG waveforms as reported in adults. The EASI lead system has been approved by the US Food and Drug Administration for assessing normal, abnormal, and paced cardiac rhythms and for detecting myocardial ischemia in patients with chest pain or silent ischemia by monitoring ST-segment elevation or depression.

Because there is individual variation between standard 12-lead ECG and EASI ECG it is

advised that clinicians only use one method when making serial ECG comparisons.

Derived precordial leads

Another approach to a reduced number of leads is to exclude certain leads from the ECG recording and instead reconstruct the excluded leads from the existing leads, using either general or patient-specific reconstruction. Nelwan and colleagues [109] studied how well the 12-lead ECG can be reconstructed from different lead subsets, always including limb leads I and II and at least one precordial lead. Patients who had unstable angina were monitored with extremity electrodes at the Mason-Likar locations and precordial leads at the standard positions. They concluded that general construction allows reconstruction of one or two precordial leads, whereas patient-specific construction allows up to four leads to be reconstructed. The best lead subset with four removed precordial leads was I, II, V2, and V5. The patient-specific reconstruction, however, presupposes a previously recorded ECG and is affected over time, probably by postural changes. In a comparison of general construction, patient-specific construction (using leads I, II, V2, and V5), and EASI leads, it was found that, when comparing ST60, patient-specific construction showed a more accurate reconstruction of the standard ECG than did the other two methods [110].

One advantage to using this system is that all limb leads and two precordial leads are known to be true leads, although the system uses more electrodes than the EASI lead system.

24-view ECG

Some investigators propose that there is no need for additional leads if the standard 12-lead ECG system could be used more effectively [15]. It is widely considered that ST elevation occurs when an injury current develops between normal and transmurally ischemic myocardium [111]. The flow of this current creates a vector toward leads with positive poles facing that area of the myocardium, seen as ST elevation. Any electrode on the other side of the heart will instead record an ST depression. For example, to see ST elevation in patients who have posterior infarcts, one would have to apply posterior thoracic electrodes (as discussed previously) or to produce a 24-view ECG in which displays of negative views of all standard leads are regarded as well (Fig. 5).

The classical standard 12-lead display includes two subsets of limb leads (I, II, III, and aVL, aVR, aVF) and the chest leads V1 to V6. It is, however, equally possible to regroup the limb leads into an orderly sequence [112]. This sequence displays the cardiac electrical activity in the frontal plane in a logical order: aVL, I, -aVR, II, and aVF, III, with each separated by

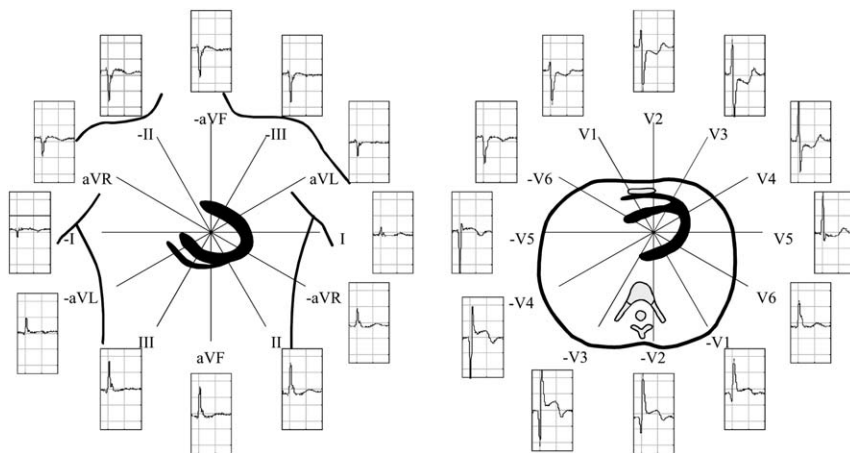


Fig. 5. The 24 views of the standard 12-lead ECG are shown as recorded from a patient receiving angioplasty balloon occlusion in a nondominant left circumflex coronary artery. QRS complexes and T waves from single cardiac cycles recorded simultaneously in the six frontal plane leads viewed from the front (*left*) and the six transverse plane leads viewed from below (*right*) are arranged like numbers around a clock face. (From: Pahlm-Webb U, Pahlm O, Sadanandan S, et al. A new method for using the direction of ST-segment deviation to localize the site of acute coronary occlusion: the 24-view standard electrocardiogram. *Am J Med* 2002;113(1):75–8; with permission.)

30°. This sequence could be enlarged further to include all inverted leads in the frontal plane, just as $-aVR$ has been inverted in the orderly sequence. In the same manner, an inversion of the precordial leads V1 to V6 could also be used. Thus, an ST depression in lead V1 would present as an ST elevation in lead $-V1$, just as an ST depression in lead aVR would present as an ST elevation in lead $-aVR$. The 24-view ECG does not require the placement of additional electrodes, because all information is available in the 12-lead ECG. An alternative of the 24-view ECG is simply to allow ST deviation (elevation or depression) in the standard 12-lead ECG to be diagnostic of acute MI. Further studies are needed, however, before any ECG diagnostic guidelines can be changed.

Summary

There is no single right answer to the question of how many leads are needed in clinical electrocardiography. This question must be answered in the context of the clinical problem to be solved. The standard 12-lead ECG is so well established that alternative lead systems must prove their advantage through well-conducted clinical studies to achieve clinical acceptance. Certain additional leads seem to add valuable information in specific patient groups. The use of a large number of leads (such as in body surface potential mapping) may add clinically relevant information; however, it is still cumbersome, and its clinical advantage is yet to be proven. Reduced lead sets emulate the 12-lead ECG reasonably well and are especially advantageous in the emergency situation.

References

- [1] Waller A. A demonstration on man of electromotive changes accompanying the hearts beat. *J Physiol* 1887;8:229–34.
- [2] Einthoven W. The different forms of the human electrocardiogram and their signification. *Lancet* 1912;1:853–61 [reprinted in *Am Heart J* 1950;40:195–211].
- [3] Wilson F, MacLeod A, Barker P. The potential variations produced by the heart beat at the apices of Einthovens triangle. *Am Heart J* 1931;7:207–11.
- [4] Wilson F, MacLeod A, Barker P, et al. The electrocardiogram in myocardial infarction with particular reference to the initial deflections of the ventricular complex. *Heart* 1933;16:155–99.
- [5] Committee of the Cardiac Society of Great Britain and Ireland and Committee of the American Heart Association. Standardisation of precordial leads. *Lancet* 1938; 221.
- [6] Barnes A, Pardee H, White P, et al. Standardization of precordial leads. *Am Heart J* 1938;15:107–8.
- [7] Barnes A, Pardee H, White P, et al. Standardization of precordial leads: supplementary report. *Am Heart J* 1938;15:235–9.
- [8] Goldberger E. A simple, indifferent, electrocardiographic electrode of zero potential and a technique of obtaining augmented, unipolar, extremity leads. *Am Heart J* 1942;23:483–92.
- [9] Mason RE, Likar I. A new system of multiple-lead exercise electrocardiography. *Am Heart J* 1966; 71(2):196–205.
- [10] Papouchado M, Walker PR, James MA, et al. Fundamental differences between the standard 12-lead electrocardiography and the modified (Mason-Likar) exercise lead system. *Eur Heart J* 1987; 8(7):725–33.
- [11] Sevilla DC, Dohrmann ML, Somelofski CA, et al. Invalidation of the resting electrocardiogram obtained via exercise electrode sites as a standard 12-lead recording. *Am J Cardiol* 1989;63(1):35–9.
- [12] Speake D, Terry P. Towards evidence based emergency medicine: best BETs from the Manchester Royal Infirmary. First ECG in chest pain. *Emerg Med J* 2001;18(1):61–2.
- [13] Blanke H, Cohen M, Schluter GU, et al. Electrocardiographic and coronary arteriographic correlations during acute myocardial infarction. *Am J Cardiol* 1984;54(3):249–55.
- [14] Saetre HA, Selvester RH, Solomon JC, et al. 16-lead ECG changes with coronary angioplasty. Location of ST-T changes with balloon occlusion of five arterial perfusion beds. *J Electrocardiol* 1992;24(Suppl):153–62.
- [15] Pahlm-Webb U, Pahlm O, Sadanandan S, et al. A new method for using the direction of ST-segment deviation to localize the site of acute coronary occlusion: the 24-view standard electrocardiogram. *Am J Med* 2002;113(1):75–8.
- [16] Brady WJ. Acute posterior wall myocardial infarction: electrocardiographic manifestations. *Am J Emerg Med* 1998;16(4):409–13.
- [17] The Joint European Society of Cardiology/American College of Cardiology Committee. Myocardial infarction redefined – A consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocardial Infarction. *Eur Heart J* 2000;21:1502–13.
- [18] Khaw K, Moreyra AE, Tannenbaum AK, et al. Improved detection of posterior myocardial wall ischemia with the 15-lead electrocardiogram. *Am Heart J* 1999;138(5 Pt 1):934–40.
- [19] Matetzky S, Freimark D, Feinberg MS, et al. Acute myocardial infarction with isolated ST-segment elevation in posterior chest leads V7–9: “hidden” ST-segment elevations revealing acute posterior infarction. *J Am Coll Cardiol* 1999; 34(3):748–53.

- [20] Oraini S, Maleki M, Tavakolian AA, et al. Prevalence and outcome of ST-segment elevation in posterior electrocardiographic leads during acute myocardial infarction. *J Electrocardiol* 1999; 32(3):275–8.
- [21] Matetzky S, Freimark D, Chouraqui P, et al. Significance of ST segment elevations in posterior chest leads (V7 to V9) in patients with acute inferior myocardial infarction: application for thrombolytic therapy. *J Am Coll Cardiol* 1998;31(3):506–11.
- [22] Chia BL, Tan HC, Yip JW, et al. Electrocardiographic patterns in posterior chest leads (V7, V8, V9) in normal subjects. *Am J Cardiol* 2000;85(7): 911–2.
- [23] Kulkarni AU, Brown R, Ayoubi M, et al. Clinical use of posterior electrocardiographic leads: a prospective electrocardiographic analysis during coronary occlusion. *Am Heart J* 1996;131(4):736–41.
- [24] Agarwal JB. Routine use of a 15-lead electrocardiogram for patients presenting to the emergency department with chest pain. *J Electrocardiol* 1998; 31(Suppl):172–7.
- [25] Agarwal JB, Khaw K, Aurignac F, et al. Importance of posterior chest leads in patients with suspected myocardial infarction, but nondiagnostic, routine 12-lead electrocardiogram. *Am J Cardiol* 1999;83(3):323–6.
- [26] Zalenski RJ, Cooke D, Rydman R, et al. Assessing the diagnostic value of an ECG containing leads V4R, V8, and V9: the 15-lead ECG. *Ann Emerg Med* 1993;22(5):786–93.
- [27] Zalenski RJ, Rydman RJ, Sloan EP, et al. Value of posterior and right ventricular leads in comparison to the standard 12-lead electrocardiogram in evaluation of ST-segment elevation in suspected acute myocardial infarction. *Am J Cardiol* 1997;79(12): 1579–85.
- [28] Ganim RP, Lewis WR, Diercks DB, et al. Right precordial and posterior electrocardiographic leads do not increase detection of ischemia in low-risk patients presenting with chest pain. *Cardiology* 2004;102(2):100–3.
- [29] Wung SF, Drew BJ. New electrocardiographic criteria for posterior wall acute myocardial ischemia validated by a percutaneous transluminal coronary angioplasty model of acute myocardial infarction. *Am J Cardiol* 2001;87(8):970–4.
- [30] Andersen HR, Falk E, Nielsen D. Right ventricular infarction: frequency, size and topography in coronary heart disease: a prospective study comprising 107 consecutive autopsies from a coronary care unit. *J Am Coll Cardiol* 1987;10(6):1223–32.
- [31] Haji SA, Movahed A. Right ventricular infarction—diagnosis and treatment. *Clin Cardiol* 2000;23(7): 473–82.
- [32] Correale E, Battista R, Martone A, et al. Electrocardiographic patterns in acute inferior myocardial infarction with and without right ventricle involvement: classification, diagnostic and prognostic value, masking effect. *Clin Cardiol* 1999;22(1): 37–44.
- [33] Andersen HR, Nielsen D, Lund O, et al. Prognostic significance of right ventricular infarction diagnosed by ST elevation in right chest leads V3R to V7R. *Int J Cardiol* 1989;23(3):349–56.
- [34] Braat SH, Brugada P, De Zwaan C, et al. Right and left ventricular ejection fraction in acute inferior wall infarction with or without ST segment elevation in lead V4R. *J Am Coll Cardiol* 1984;4(5): 940–4.
- [35] Braat SH, de Zwaan C, Brugada P, et al. Right ventricular involvement with acute inferior wall myocardial infarction identifies high risk of developing atrioventricular nodal conduction disturbances. *Am Heart J* 1984;107(6):1183–7.
- [36] Chou TC, Van der Bel-Kahn J, Allen J, et al. Electrocardiographic diagnosis of right ventricular infarction. *Am J Med* 1981;70(6):1175–80.
- [37] Croft CH, Nicod P, Corbett JR, et al. Detection of acute right ventricular infarction by right precordial electrocardiography. *Am J Cardiol* 1982;50(3): 421–7.
- [38] Braat SH, Brugada P, de Zwaan C, et al. Value of electrocardiogram in diagnosing right ventricular involvement in patients with an acute inferior wall myocardial infarction. *Br Heart J* 1983;49(4): 368–72.
- [39] Lopez-Sendon J, Coma-Canella I, Alcasena S, et al. Electrocardiographic findings in acute right ventricular infarction: sensitivity and specificity of electrocardiographic alterations in right precordial leads V4R, V3R, V1, V2, and V3. *J Am Coll Cardiol* 1985;6(6):1273–9.
- [40] Andersen HR, Thomsen PE, Nielsen TT, et al. ST deviation in right chest leads V3R to V7R during percutaneous transluminal coronary angioplasty. *Am Heart J* 1990;119(3 Pt 1):490–3.
- [41] Morgera T, Alberti E, Silvestri F, et al. Right precordial ST and QRS changes in the diagnosis of right ventricular infarction. *Am Heart J* 1984; 108(1):13–8.
- [42] Andersen HR, Nielsen D, Hansen LG. The normal right chest electrocardiogram. *J Electrocardiol* 1987;20(1):27–32.
- [43] Braat SH, Brugada P, den Dulk K, et al. Value of lead V4R for recognition of the infarct coronary artery in acute inferior myocardial infarction. *Am J Cardiol* 1984;53(11):1538–41.
- [44] Ueshima K, Kobayashi N, Kamata J, et al. Do the right precordial leads during exercise testing contribute to detection of coronary artery disease? *Clin Cardiol* 2004;27(2):101–5.
- [45] Shry EA, Eckart RE, Furgerson JL, et al. Addition of right-sided and posterior precordial leads during stress testing. *Am Heart J* 2003;146(6):1090–4.
- [46] Michaelides AP, Psomadaki ZD, Dilaveris PE, et al. Improved detection of coronary artery disease by exercise electrocardiography with the use of

- right precordial leads. *N Engl J Med* 1999;340(5):340–5.
- [47] Akula R, Hasan SP, Alhassen M, et al. Right-sided EKG in pulmonary embolism. *J Natl Med Assoc* 2003;95(8):714–7.
- [48] Chia BL, Tan HC, Lim YT. Right sided chest lead electrocardiographic abnormalities in acute pulmonary embolism. *Int J Cardiol* 1997;61(1):43–6.
- [49] Waller AD. On the electromotive changes connected with the beat of the mammalian heart, and of the human heart in particular. *Philos Trans R Soc* 1889;180:169–94.
- [50] Taccardi B. Distribution of heart potentials on the thoracic surface of normal human subjects. *Circ Res* 1963;12:341–52.
- [51] Nahum LH, Mauro A, Chernoff HM, et al. Instantaneous equipotential distribution on surface of the human body for various instants in the cardiac cycle. *J Appl Physiol* 1951;3(8):454–64.
- [52] Gulrajani RM, Roberge FA, Mailloux GE. The forward problem of electrocardiography. In: Macfarlane P, Lawrie T, editors. *Comprehensive electrocardiology: theory and practice in health and disease*. New York: Pergamon Press; 1989. p. 197–236.
- [53] Gulrajani RM, Roberge FA, Savard P. The inverse problem of electrocardiography. In: Macfarlane P, Lawrie T, editors. *Comprehensive electrocardiology: theory and practice in health and disease*. New York: Pergamon Press; 1989. p. 237–88.
- [54] Montague TJ, Smith ER, Spencer CA, et al. Body surface electrocardiographic mapping in inferior myocardial infarction. Manifestation of left and right ventricular involvement. *Circulation* 1983;67(3):665–73.
- [55] Kornreich F, Rautaharju PM, Warren J, et al. Identification of best electrocardiographic leads for diagnosing myocardial infarction by statistical analysis of body surface potential maps. *Am J Cardiol* 1985;56(13):852–6.
- [56] Menown IB, Patterson RS, MacKenzie G, et al. Body-surface map models for early diagnosis of acute myocardial infarction. *J Electrocardiol* 1998;31(Suppl):180–8.
- [57] Menown IB, Allen J, Anderson JM, et al. Early diagnosis of right ventricular or posterior infarction associated with inferior wall left ventricular acute myocardial infarction. *Am J Cardiol* 2000;85(8):934–8.
- [58] Maynard SJ, Menown IB, Manoharan G, et al. Body surface mapping improves early diagnosis of acute myocardial infarction in patients with chest pain and left bundle branch block. *Heart* 2003;89(9):998–1002.
- [59] Yamada K, Toyama J, Sugeno J, et al. Body surface isopotential maps. Clinical application to the diagnosis of myocardial infarction. *Jpn Heart J* 1978;19(1):28–45.
- [60] Vincent GM, Abildskov JA, Burgess MJ, et al. Diagnosis of old inferior myocardial infarction by body surface isopotential mapping. *Am J Cardiol* 1977;39(4):510–5.
- [61] Vesterinen P, Hanninen H, Karvonen M, et al. Temporal analysis of the depolarization wave of healed myocardial infarction in body surface potential mapping. *Ann Noninvasive Electrocardiol* 2004;9(3):234–42.
- [62] Toyama S, Suzuki K, Koyama M, et al. The body surface isopotential mapping of the QRS wave in myocardial infarction. a comparative study of the scintigram with thallium-201. *J Electrocardiol* 1982;15(3):241–8.
- [63] Toyama S, Suzuki K, Koyama M. The isopotential mapping of the T wave in myocardial infarction. *J Electrocardiol* 1980;13(4):323–30.
- [64] Pham-Huy H, Gulrajani RM, Roberge FA, et al. A comparative evaluation of three different approaches for detecting body surface isopotential map abnormalities in patients with myocardial infarction. *J Electrocardiol* 1981;14(1):43–55.
- [65] Osugi J, Ohta T, Toyama J, et al. Body surface isopotential maps in old inferior myocardial infarction undetectable by 12 lead electrocardiogram. *J Electrocardiol* 1984;17(1):55–62.
- [66] Ohta T, Toyama J, Ohsugi J, et al. Correlation between body surface isopotential maps and left ventriculograms in patients with old anterior myocardial infarction. *Jpn Heart J* 1981;22(5):747–61.
- [67] Ohta T, Kinoshita A, Ohsugi J, et al. Correlation between body surface isopotential maps and left ventriculograms in patients with old inferoposterior myocardial infarction. *Am Heart J* 1982;104(6):1262–70.
- [68] Medvegy M, Preda I, Savard P, et al. New body surface isopotential map evaluation method to detect minor potential losses in non-Q-wave myocardial infarction. *Circulation* 2000;101(10):1115–21.
- [69] Kornreich F, Montague TJ, Rautaharju PM. Identification of first acute Q wave and non-Q wave myocardial infarction by multivariate analysis of body surface potential maps. *Circulation* 1991;84(6):2442–53.
- [70] Hirai M, Ohta T, Kinoshita A, et al. Body surface isopotential maps in old anterior myocardial infarction undetectable by 12-lead electrocardiograms. *Am Heart J* 1984;108(4 Pt 1):975–82.
- [71] Flowers NC, Horan LG, Sohi GS, et al. New evidence for inferoposterior myocardial infarction on surface potential maps. *Am J Cardiol* 1976;38(5):576–81.
- [72] Flowers NC, Horan LG, Johnson JC. Anterior infarctional changes occurring during mid and late ventricular activation detectable by surface mapping techniques. *Circulation* 1976;54(6):906–13.
- [73] De Ambroggi L, Bertoni T, Rabbia C, et al. Body surface potential maps in old inferior myocardial

- infarction. Assessment of diagnostic criteria. *J Electrocardiol* 1986;19(3):225–34.
- [74] Ackaoui A, Nadeau R, Sestier F, et al. Myocardial infarction diagnosis with body surface potential mapping, electrocardiography, vectorcardiography and thallium-201 scintigraphy: a correlative study with left ventriculography. *Clin Invest Med* 1985;8(1):68–77.
 - [75] Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. *N Engl J Med* 1996;334(8):481–7.
 - [76] Hands ME, Cook EF, Stone PH, et al. Electrocardiographic diagnosis of myocardial infarction in the presence of complete left bundle branch block. *Am Heart J* 1988;116(1 Pt 1):23–31.
 - [77] Menown IB, Allen J, Anderson JM, et al. Noninvasive assessment of reperfusion after fibrinolytic therapy for acute myocardial infarction. *Am J Cardiol* 2000;86(7):736–41.
 - [78] Cahyadi YH, Takekoshi N, Matsui S. Clinical efficacy of PTCA and identification of restenosis: evaluation by serial body surface potential mapping. *Am Heart J* 1991;121(4 Pt 1):1080–7.
 - [79] Manninen H, Takala P, Makijarvi M, et al. Recording locations in multichannel magnetocardiography and body surface potential mapping sensitive for regional exercise-induced myocardial ischemia. *Basic Res Cardiol* 2001;96(4):405–14.
 - [80] Montague TJ, Johnstone DE, Spencer CA, et al. Body surface potential maps with low-level exercise in isolated left anterior descending coronary artery disease. *Am J Cardiol* 1988;61(4):273–82.
 - [81] Boudik F, Anger Z, Aschermann M, et al. Dipyrindamole body surface potential mapping: noninvasive differentiation of syndrome X from coronary artery disease. *J Electrocardiol* 2002;35(3):181–91.
 - [82] Kornreich F, Montague TJ, Rautaharju PM, et al. Identification of best electrocardiographic leads for diagnosing left ventricular hypertrophy by statistical analysis of body surface potential maps. *Am J Cardiol* 1988;62(17):1285–91.
 - [83] Kornreich F, Montague TJ, van Herpen G, et al. Improved prediction of left ventricular mass by regression analysis of body surface potential maps. *Am J Cardiol* 1990;66(4):485–92.
 - [84] Holt JH Jr, Barnard AC, Kramer JO Jr. Multiple dipole electrocardiography: a comparison of electrically and angiographically determined left ventricular masses. *Circulation* 1978;57(6):1129–33.
 - [85] Yamada K, Toyama J, Wada M, et al. Body surface isopotential mapping in Wolff-Parkinson-White syndrome: noninvasive method to determine the localization of the accessory atrioventricular pathway. *Am Heart J* 1975;90(6):721–34.
 - [86] Tseng YZ, Hsu KL, Chiang FT, et al. The use of body surface potential map for identifying sites of accessory pathway in patients with Wolff-Parkinson-White syndrome. *Jpn Heart J* 1998;39(4):445–55.
 - [87] Liebman J, Zeno JA, Olshansky B, et al. Electrocardiographic body surface potential mapping in the Wolff-Parkinson-White syndrome. Noninvasive determination of the ventricular insertion sites of accessory atrioventricular connections. *Circulation* 1991;83(3):886–901.
 - [88] Iwa T, Magara T. Correlation between localization of accessory conduction pathway and body surface maps in the Wolff-Parkinson-White syndrome. *Jpn Circ J* 1981;45(10):1192–8.
 - [89] Dubuc M, Nadeau R, Tremblay G, et al. Pace mapping using body surface potential maps to guide catheter ablation of accessory pathways in patients with Wolff-Parkinson-White syndrome. *Circulation* 1993;87(1):135–43.
 - [90] De Ambroggi L, Taccardi B, Macchi E. Body-surface maps of heart potentials: tentative localization of pre-excited areas in forty-two Wolff-Parkinson-White patients. *Circulation* 1976;54(2):251–63.
 - [91] Benson DW Jr, Sterba R, Gallagher JJ, et al. Localization of the site of ventricular preexcitation with body surface maps in patients with Wolff-Parkinson-White syndrome. *Circulation* 1982;65(6):1259–68.
 - [92] Carley SD, Jenkins M, Jones KM. Body surface mapping versus the standard 12 lead ECG in the detection of myocardial infarction amongst emergency department patients: a Bayesian approach. *Resuscitation* 2005;64(3):309–14.
 - [93] Frank E. An accurate, clinically practical system for spatial vectorcardiography. *Circulation* 1954;13(4):737–49.
 - [94] Dellborg M, Topol EJ, Swedberg K. Dynamic QRS complex and ST segment vectorcardiographic monitoring can identify vessel patency in patients with acute myocardial infarction treated with reperfusion therapy. *Am Heart J* 1991;122(4 Pt 1):943–8.
 - [95] Dellborg M, Steg P, Simoons M, et al. Vectorcardiographic monitoring to assess early vessel patency after reperfusion therapy for acute myocardial infarction. *Eur Heart J* 1995;16(1):21–9.
 - [96] Dellborg M, Herlitz J, Risenfors M, et al. Electrocardiographic assessment of infarct size: comparison between QRS scoring of 12-lead electrocardiography and dynamic vectorcardiography. *Int J Cardiol* 1993;40(2):167–72.
 - [97] Willems JL, Abreu-Lima C, Arnaud P, et al. Assessment of the diagnostic performance of ECG computer programs and cardiologists. In: Common standards for quantitative electrocardiography.

- CSE 10th progress report. Leuven (Belgium): Acco; 1990. p. 197–236.
- [98] Dower GE, Yakush A, Nazzal SB, et al. Deriving the 12-lead electrocardiogram from four (EASI) electrodes. *J Electrocardiol* 1988;21(Suppl): S182–7.
 - [99] Feild DQ, Feldman CL, Horacek BM. Improved EASI coefficients: their derivation, values, and performance. *J Electrocardiol* 2002;35(Suppl):23–33.
 - [100] Klein M, Key-Brothers I, Feldman C. Can the vectorcardiographically derived EASI ECG be a suitable surrogate for the standard ECG in selected circumstances? *Proc Comput Cardiol* 1997;5(3): 721–4.
 - [101] Drew BJ, Pelter MM, Wung SF, et al. Accuracy of the EASI 12-lead electrocardiogram compared to the standard 12-lead electrocardiogram for diagnosing multiple cardiac abnormalities. *J Electrocardiol* 1999;32(Suppl):38–47.
 - [102] Drew BJ, Adams MG, Pelter MM, et al. ST segment monitoring with a derived 12-lead electrocardiogram is superior to routine cardiac care unit monitoring. *Am J Crit Care* 1996;5(3): 198–206.
 - [103] Rautaharju PM, Zhou SH, Hancock EW, et al. Comparability of 12-lead ECGs derived from EASI leads with standard 12-lead ECGs in the classification of acute myocardial ischemia and old myocardial infarction. *J Electrocardiol* 2002; 35(Suppl):35–9.
 - [104] Feldman CL, MacCallum G, Hartley LH. Comparison of the standard ECG with the EASICardiogram for ischemia detection during exercise monitoring. *Proc Comput Cardiol* 1997;24: 343–5.
 - [105] Wehr G, Peters R, Khalife K, et al. A vector-based 5 electrode 12-lead ECG (EASI) is equivalent to the conventional 12-lead ECG for diagnosis of myocardial ischemia. *J Am Coll Cardiol* 2002; 39(Suppl):122A.
 - [106] Horacek BM, Warren JW, et al. Diagnostic accuracy of derived versus standard 12-lead electrocardiograms. *J Electrocardiol* 2000;33(Suppl):155–60.
 - [107] Sejersten M, Pahlm O, Pettersson J, et al. The relative accuracies of ECG precordial lead waveforms derived from EASI leads and those acquired from paramedic applied standard leads. *J Electrocardiol* 2003;36(3):179–85.
 - [108] Pahlm O, Pettersson J, Thulin A, et al. Comparison of waveforms in conventional 12-lead ECGs and those derived from EASI leads in children. *J Electrocardiol* 2003;36(1):25–31.
 - [109] Nelwan SP, Kors JA, Meij SH, et al. Reconstruction of the 12-lead electrocardiogram from reduced lead sets. *J Electrocardiol* 2004;37(1):11–8.
 - [110] Nelwan SP, Crater SW, Meij SH, et al. Simultaneous comparison of three derived 12-lead ECGs with standard ECG at rest and during percutaneous coronary occlusion. *J Electrocardiol* 2004; 37(Suppl):171–2.
 - [111] Wagner GS. Marriot's practical electrocardiography. 10th edition. Philadelphia: Lippincott Williams & Wilkins; 2001.
 - [112] Anderson ST, Pahlm O, Selvester RH, et al. Panoramic display of the orderly sequenced 12-lead ECG. *J Electrocardiol* 1994;27(4):347–52.