

Comparative Accuracy of Electrocardiographic and Vectorcardiographic Criteria for Inferior Myocardial Infarction

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SUMMARY Numerous criteria for the diagnosis of inferior wall myocardial infarction by electrocardiogram (ECG) and vectorcardiogram (VCG) have been published, but they have not been subjected to a systematic, independent evaluation. Accordingly, we studied 146 patients undergoing cardiac catheterization; 63 were normal and 83 had a history of infarction, a significant right coronary lesion and an inferior wall motion abnormality (inferior infarction group). No ECG or VCG criteria were considered in the designation of the two groups; rather, three sets of ECG and VCG criteria were evaluated for this purpose. Specificity was excellent (98–100%) and sensitivity was poor (4–34%) by all three sets of ECG criteria, but the 1949 ECG criteria of Meyers et al. are the least sensitive (4%, $p < 0.001$). Specificity (90–100%) and sensitivity (82–84%) were very good by all three VCG criteria. The VCG criteria of Starr et al. gave no false-positive results in our normal group. Because of enhanced sensitivity, the overall accuracy of the VCG was higher than that of the ECG for the diagnosis of inferior infarction (90% vs 62%, $p < 0.001$). We conclude that more recent ECG criteria for the diagnosis of inferior wall myocardial infarction are highly specific, but insensitive compared with VCG criteria.

THE NONINVASIVE, premortem diagnosis of myocardial infarction is important for proper patient evaluation and treatment. The presence of diagnostic Q waves on the electrocardiogram (ECG) is considered strong evidence of previous myocardial damage and their absence is used to refute this diagnosis. The ECG criteria for infarction were developed more than 20 years ago, before modern standardization of ECG response characteristics, and before the availability of sensitive methods for diagnosing myocardial infarction. More recently, the vectorcardiogram (VCG) has been advanced as a diagnostic tool which is superior to the ECG for the diagnosis of infarction. Numerous investigators have published criteria for the diagnosis of inferior wall myocardial infarction by ECG and VCG.¹⁻⁹ However, these criteria, especially those of the VCG, have not been subjected to a systematic, independent evaluation. Therefore, the objective of this study was to compare and evaluate prospectively the sensitivity and specificity of published ECG and VCG criteria for detecting inferior wall myocardial infarction defined by invasive methods.

Methods

Patients

The study population consisted of 146 consecutive patients who underwent cardiac catheterization and

met criteria for one of two groups. Group 1, the normal group, consisted of 63 patients with normal right- and left-heart pressures, cardiac output, left ventricular wall motion, ejection fraction, valvular function and coronary arteries. Sixteen of these 63 were evaluated for atypical chest pain and 47, whose job required the exclusion of coronary artery disease, were evaluated because of an abnormal screening ECG exercise test. Group 2, the inferior infarction group, consisted of 83 patients with a history of myocardial infarction, defined as a period of prolonged chest pain accompanied by a rise and fall in serum creatine kinase-MB isoenzyme. No ECG or VCG findings were required for the diagnosis, but patients with left bundle branch block were excluded from the study. Instead, evidence of critical narrowing of a dominant right or left circumflex coronary artery on selective coronary cineangiography and an inferior wall motion abnormality on a 30° right anterior oblique left ventricular cineangiogram were required. These inferior infarction patients were subgrouped according to the degree of inferior wall motion abnormality. Group 2A ($n = 26$) consisted of patients with inferior wall hypokinesis; Group 2B ($n = 57$) consisted of patients with inferior wall akinesis or dyskinesis.

Angiography

Each patient underwent a complete diagnostic right- and left-heart catheterization with left ventriculography in the 30° right anterior oblique projection and selective right and left coronary artery cineangiography in multiple projections by the standard Sones¹⁰ or Judkins¹¹ technique. Intraluminal coronary arterial obstruction was measured in each artery and considered to be critical if the diameter narrowing was 70% or greater. Right or left coronary artery dominance was determined by which artery supplied the posterior descending coronary artery.

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Left ventricular cineangiography was used to evaluate the left ventricular contraction pattern. The left ventricular end-diastolic frame of a sinus beat was taken as the initial position and frame-by-frame analysis was performed to end-systole to divide the patients into three groups: (1) normal wall motion, (2) inferior wall hypokinesis and (3) inferior wall akinesis or dyskinesis.¹² All cineangiograms were reviewed by at least two observers who had not seen the ECG or the VCG of the patients. Disagreements were resolved by rereview with another observer.

Electrocardiogram and Vectorcardiogram

Within 48 hours before cardiac catheterization, an ECG and a VCG were obtained from each patient. The ECG was recorded on either a Marquette Series 3000 or a Cambridge 3038/2, three-channel electrocardiograph at a paper speed of 25 mm/sec, with standardization adjusted to 10 mm/mV. The VCG was obtained using the Frank lead placement and was recorded in the frontal, horizontal and right sagittal planes on an Instrument for Cardiac Research Model VCG-2T vectorcardiograph with the QRS loops recorded at 40 mm/mV and 80 mm/mV. Time marks were recorded each 2.5 msec. The ECG and VCG of each patient were evaluated without knowledge of the historical or angiographic data. In each case, a decision was made as to the presence or absence of an inferior wall myocardial infarction as predicted by the published criteria.

The following ECG criteria were evaluated: The ECG was positive by the criteria of Myers et al.³ when, using lead aV_F, the QRS magnitude was at least 0.5 mV and a Q wave was present that had a duration of at least 0.03 second from onset to nadir, and the Q:R ratio was at least 1:4. If the QRS magnitude was less than 0.5 mV or if Q:R was < 1:4, the Q wave must have been at least 0.04 second to the nadir. The ECG was positive by the criteria of Walsh et al.⁷ if lead III had a Q wave of at least 0.04 second duration and a Q:R of > 1:4, or if lead aV_F had a Q wave meeting either of the criteria for lead III. The criteria for a positive ECG by the New York Heart Association (NYHA)⁹ were a Q wave at least 0.03 second in duration and a Q:R at least 1:4 in leads III and aV_F.

The VCG criteria evaluated are as follows: The VCG criteria of Young and Williams⁸ involved the frontal plane (QRS) loop. They required an early superior force with clockwise (CW) rotation, at least 20 msec in duration and an X-axis intercept > 0.25 mV from the origin. If the early superior force was almost CW, then it must have been at least 25 msec in duration. The early CW superior force might be of any duration if the angle of the maximum QRS vector was above 10°. If the initial force was inferior, it must have been to the right, completely CW, and followed by CW superior forces which were at least 25 msec in duration and 0.25 mV in magnitude. If the angle of the maximum QRS vector was above 10°, the superior forces need not meet the duration and magnitude criteria. If an anterior infarction is present, then the

initial forces could be to the left and counterclockwise, if subsequent forces are superior. The criteria of Starr et al.⁵ also pertained to the frontal plane QRS loop and included both contour and spatial components. The contour components were that the early forces must be directed superiorly and CW or, if inferior, then inferiorly displaced less than 10 msec and must cross the X-axis to the right. In addition, one spatial criterion must be met. The spatial criteria were as follows: (1) the superior forces must be at least 25 msec in duration and cross the X-axis at least 0.30 mV to the left of the origin; (2) a maximum QRS vector above 15°; and (3) a maximum superior force of at least 0.1 mV and a ratio of maximum superior force to maximum inferior force of at least 1:5.

The criteria of Stein and Simon⁶ included one of the following: (1) 20-msec QRS vector in the right sagittal plane superior to -45°, or (2) a frontal plane finding of (origin to X-intercept voltage)/(maximum QRS voltage) greater than 0.22. Each set of criteria is cited in the text by the name of the first author of the report.

Data Analysis

Statistical analysis was performed using the chi-square test with Yates' correction for continuity.¹³ Multiple tests were performed to identify differences between sets of criteria, so the statistical limits of significance were adjusted to avoid a type I error.¹⁴ Therefore, for these comparisons, it was determined that a *p* value < 0.01 would indicate significance. We also evaluated each set of criteria for sensitivity, specificity and overall test accuracy.¹⁵

Results

Electrocardiogram

The number of positive inferior infarction diagnoses by each set of ECG criteria is shown in table 1. Specificity was excellent and sensitivity was poor by all three sets of ECG criteria. However, Myers's criteria were significantly less sensitive for the diagnosis of inferior infarction than either the Walsh or the NYHA criteria (*p* < 0.001). Overall test accuracy of the ECG for the presence or absence of inferior infarction was 45% for the Myers criteria and 62% for both the Walsh and NYHA (*p* < 0.01). In comparing group 2A to group 2B, there was no signifi-

TABLE 1. Number of Positive Diagnoses of Inferior Infarction by Electrocardiographic Criteria

Group	n	Criteria of					
		Myers	%	Walsh	%	NYHA	%
1	63	0	100	1	98	0	100
2	83	3	4	28	34	28	34
2A	26	0	0	5	19	6	23
2B	57	3	5	23	40	22	39

The percentages indicate the incidence of true-negative diagnoses (specificity) for group 1 and true-positive diagnoses (sensitivity) for group 2.

cant increase in sensitivity as dyssynergy increased for any of the ECG criteria.

Vectorcardiogram

A comparison of the various VCG criteria is shown in table 2. Specificity was excellent by all three sets of VCG criteria. However, only the criteria by Starr yielded no false-positive results. Sensitivity was good by all three sets of criteria. The overall accuracy of the VCG was 90% for the Starr criteria and 88% for both the Young and Stein criteria. Six group 2 patients had VCG evidence of left ventricular hypertrophy (maximum QRS, transverse plane > 1.8 mV), but this did not influence the results. Five patients in group 2 also had anterior wall abnormalities and VCG evidence of anterior infarction, but all were positive by the VCG criteria of Starr and those of Young.

VCG criteria have both spatial and contour components. To evaluate the necessity of including the contour components, we measured the frequency of infarction diagnosis using only the spatial criterion of initial superior forces lasting at least 25 msec as diagnostic of inferior infarction. The results are shown in table 3. Spatial criteria alone, when compared to Starr's combined spatial and contour criteria, were significantly less specific (84% vs 100%, $p < 0.005$), and had an overall accuracy of only 80%.

ECG and VCG Comparison

The criteria of the NYHA were the only ECG criteria with both no false positives and with a sensitivity equal to or greater than the other ECG criteria. Also, the VCG criteria of Starr had no false

TABLE 2. Number of Positive Diagnoses of Inferior Infarction by Vectorcardiographic Criteria

Group	n	Criteria of					
		Young	%	Starr	%	Stein	%
1	63	5	90	0	100	3	95
2	83	70	84	68	82	68	82
2A	26	23	88	20	77	22	85
2B	57	47	82	48	84	46	81

The percentages indicate the incidence of true-negative diagnoses (specificity) for group 1 and true-positive diagnoses (sensitivity) for group 2.

TABLE 3. Number of Inferior Infarction Diagnoses by the Criterion of Initial Superior Vector Forces ≥ 25 Msec

Group	Positive	%
1	10	84
2	64	77
2A	18	69
2B	46	81

Percent indicates the incidence of true-negative diagnoses (specificity) for group 1 and true-positive diagnoses (sensitivity) for group 2.

TABLE 4. Comparison of Electrocardiographic Criteria by New York Heart Association and Vectorcardiographic Criteria by Starr

Group	ECG	VCG	p
1	100%	100%	NS
2	34%	82%	< 0.001
2A	23%	77%	< 0.001
2B	39%	84%	< 0.001

Percent indicates the incidence of true-negative diagnoses (specificity) for group 1 and true-positive diagnoses (sensitivity) for group 2.

positives and were as sensitive as the other VCG criteria. Therefore, further comparison was made between the ECG criteria of the NYHA and the VCG criteria of Starr (table 4). Both methods were highly specific for the diagnosis of inferior infarction, but the VCG criteria of Starr were more sensitive than the NYHA ECG criteria (82% vs 34%, $p < 0.001$). In addition, because the VCG was more sensitive, the overall accuracy of the VCG was significantly greater than for the ECG (90% vs 62%, $p < 0.001$). Furthermore, in no patient with infarction was the ECG positive and the VCG negative. However, many patients with inferior infarction were detected by the VCG, but not by the ECG.

Discussion

We prospectively evaluated ECG and VCG findings in 63 normal subjects and 83 patients with inferior wall myocardial infarction established by history, cardiac enzymes and cardiac catheterization. The original ECG criteria of Myers et al.⁸ published in 1949 were found to be specific, but insensitive, indicators of the presence of inferior infarction. The more recent criteria of Walsh et al.⁷ and of the NYHA⁹ are more sensitive and are also highly specific for the diagnosis of inferior infarction. In comparison, the VCG is much more sensitive to the presence of an inferior infarction, and the criteria of Starr and his colleagues⁶ gave no false-positive results in our patients. The superior performance of the criteria of Starr is a result of a combination of both spatial and contour components, as the spatial components alone are significantly less specific for the VCG diagnosis of inferior wall myocardial infarction.

Several inherent biases have arisen in studies of the ECG and the VCG, which we have attempted to eliminate in our study. The major bias involved the selection of patient groups. In several studies, the patient group was defined as those with ECG evidence of infarction.^{7, 8, 16, 17} Thus, the patients were preselected for study by the very method which was to be evaluated. No ECG or VCG infarction criteria were required for inclusion into our study. Also, post-mortem studies preselect those patients with more severe disease and, correspondingly, larger areas of infarction.³ This would probably increase the apparent sensitivity of the ECG and the VCG. Other investi-

gators have correlated the ECG and VCG with left ventriculographic abnormalities without documenting a clinical episode of myocardial infarction.¹⁸⁻²⁶ This method may have included patients with silent infarction or inferior wall motion abnormality from chronic ischemia without infarction.

We selected our criteria for inferior myocardial infarction to avoid the above-noted biases in the previous studies. Each of our patients had historical, enzymatic and cardiac catheterization evidence of infarction. When coupled with a clinical history of infarction, the presence of significant amounts of MB-creatinine kinase in the serum is a sensitive and specific indicator of acute myocardial infarction.²⁷ This clinical presentation was accompanied by severe coronary artery obstruction and localized inferior left ventricular wall motion abnormalities on the cineangiogram in all our patients. We believe that this combination provides the best available antemortem diagnosis of inferior wall infarction.

The presence of coronary artery disease alone is not proof that infarction has occurred. Similarly, wall motion abnormalities have been demonstrated in myocardium supplied by narrowed arteries without infarction if dilatation and hypertrophy of the left ventricle are present.²⁸ Chronic ischemia could be a factor in the wall motion abnormalities in some of our patients;²⁹ however, the clinical documentation of an infarction makes this the most probable cause of the inferior wall motion abnormalities.

Other investigators have concluded that ECG criteria for inferior infarction lack appropriate sensitivity.^{1, 17, 30} When Myers et al. presented their criteria, they used postmortem specimens. The result was a description of deep, wide Q waves because their patients all had large inferior infarctions. Young and Williams⁸ evaluated 100 patients by VCG with a previous inferior infarction. Each of their patients had, at one time, satisfied the rigorous ECG criteria of Myers et al. for inferior infarction. Nevertheless, less than 50% of their patients satisfied ECG criteria for inferior infarction at the time of their study. Our results are similar to theirs in that the ECG was equal in specificity, but much less sensitive, and therefore, less accurate than the VCG for the diagnosis of inferior infarction.

Several characteristics of the 12-lead ECG are likely to result in lower accuracy of diagnosis compared with the VCG. The first characteristic is the inherent measurement error. At a paper speed of 25 mm/sec, a 40-msec displacement would occur over a distance of only 1 mm. Thus, as a practical matter, measurements are an approximation to the nearest 10 msec. In addition, it is difficult to determine accurately the onset of the QRS complex. In contrast, VCG loops are recorded at 2.5-msec intervals, making the duration of superior or inferior forces accurate to within 1 msec, a 1000% improvement over the ECG. A second characteristic of the ECG is that no contour criteria are possible because recordings are not made with reference to planes, as they are with the VCG. The importance of contour criteria is demonstrated in

table 4. If all VCGs with an initial superior force duration of at least 25 msec are considered to be positive for the diagnosis of inferior infarction, the number of false-positive diagnoses is 16% — significantly greater than that found by Starr et al. when contour, as well as spatial, criteria are required for the diagnosis. Therefore, our data suggest that the VCG is accurate and extremely valuable in detecting a prior inferior wall myocardial infarction in patients with non-diagnostic ECGs, and that this enhanced accuracy is largely a result of the addition of contour criteria.

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Usefulness of the Valsalva Maneuver in Management of the Long QT Syndrome

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SUMMARY Exercise or isoproterenol infusion may evoke ventricular arrhythmias in patients with the long QT syndrome. We examined the electrocardiographic effects of the Valsalva maneuver in eight patients with the long QT syndrome and nine healthy subjects. The Valsalva maneuver lengthened the QTc interval in both groups, but the lengthening was greater in the patients. In the patients who were having frequent attacks of ventricular tachycardia, the Valsalva-induced prolongation of the QTc interval was particularly remarkable and was associated with the development of T-wave alternans and short runs of ventricular tachycardia. Propranolol effectively suppressed the lengthening of the QTc interval during Valsalva strain and prevented Valsalva-induced ventricular arrhythmia. These results suggest that the Valsalva maneuver may be useful in evaluating the risk of ventricular tachyarrhythmias and the efficacy of drug treatment in patients with the long QT syndrome.

THE LONG QT SYNDROME is a hereditary disorder with a very high mortality.¹⁻⁶ It is characterized by a prolonged QT interval and T-wave alternans on the ECG and by frequent syncopal attacks due to ventricular fibrillation, which almost exclusively follow emotional or physical stress.¹⁻⁷

Clinical observations of the QT interval and T waves during block or stimulation of the stellate

ganglia^{4, 5, 8-12} in patients with the long QT syndrome suggests that the relative predominance of left adrenergic neural activity in the heart is the pathogenetic mechanism of the long QT syndrome.^{4, 8, 11} However, it is still unknown whether adrenergic neural asymmetry is due to function derangement of the sympathetic nervous system^{4, 8} or to cardiac focal neuritis, which may alter reception of sympathetic neural traffic.¹³

Exercise or isoproterenol infusion reportedly produces marked prolongation of the QT interval and T-wave alternans, and may precipitate ventricular tachyarrhythmias in patients with the long QT syndrome.^{4-7, 14, 15} In this study we examined whether reflex sympathetic activation by Valsalva maneuver evokes prolongation of the QT interval and precipitates ventricular tachyarrhythmias and evaluated the effect of propranolol, a drug commonly used in the treatment of this syndrome, on the Valsalva-induced prolongation of the QT interval.

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