Vectorcardiography Risk Stratifies Emergency Department Chest Pain Patients with Left Ventricular Hypertrophy on the Initial 12-Lead ECG

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Background: Vectorcardiographic (VCG) measurements of ST-vector magnitude (VM) and QRS-vector difference (VD) have been demonstrated to be independent predictors of adverse outcome (AO) and acute myocardial infarction (AMI) in emergency department (ED) chest pain patients with absence of bundle branch block or left ventricular hypertrophy (LVH) on the initial 12-lead electrocardiogram (ECG). The prognostic value of ST-VM and QRS-VD in ED chest pain patients with LVH on the initial 12-lead ECG has not been previously investigated.

Methods: A prospective observational study was performed in 196 consecutive ED chest pain patients with suspected AMI and presence of voltage criteria for LVH on initial ECG who underwent continuous VCG monitoring during the initial evaluation. The optimal baseline ST-VM value and 2-hour QRS-VD value were defined as the most accurate value on the receiver operator characteristic curve (value with lowest false-negative and false-positive rate). Thirty-day AO was defined as AMI, percutaneous coronary intervention, coronary artery bypass grafting (CABG), or cardiac death occurring within 30 days of initial ED visit.

Results: Fourteen patients (7.1%) were diagnosed as 24-hour AMI and 28 patients (14.3%) experienced 30-day AO. The optimal cut-off value for predicting 30-day AO was $>124 \mu\text{V}$ for ST-VM and $>21.7 \mu\text{V}$ for QRS-VD. Patients with either a positive ST-VM or a positive QRS-VD had 8.8 times increased odds of AMI (95% confidence interval, CI, 1.9–40.3; $P = 0.003$); 4.3 times increased odds of 30-day PTCA/CABG (95% CI 1.3–13.8; $P = 0.019$); and 3.8 times increased odds of 30-day AO (95% CI 1.6–9.3; $P = 0.003$).

Conclusions: Baseline ST-VM and 2-hour QRS-VD risk stratifies ED chest pain patients with LVH voltage criteria on the initial 12-lead ECG.

A.N.E. 2004;9(2):149–155

acute myocardial infarction; acute coronary syndromes; vectorcardiography; ST-vector magnitude; QRS-vector difference; ST-segment monitoring

The presence of left ventricular hypertrophy (LVH) as detected by the 12-lead electrocardiogram (ECG) is a powerful independent predictor of sudden death, cardiovascular disease, and cardiac failure in chest pain patients.^{1–4} Identification of the electrocardiographic injury and ischemia on the ECG in patients with LVH is of paramount importance in the identification of those whom may benefit from treatment with aspirin, heparin, beta-blockers, platelet glycoprotein IIb/IIIa inhibitors, and emer-

gent reperfusion therapy.^{5–7} However, the repolarization abnormalities accompanying LVH can make interpretation of injury, ischemia, and infarction difficult as LVH patients will have ≥ 1 mm ST-segment elevation in leads with a predominant negative QRS complex, ST-depressions, and T-wave inversions in leads with a predominant positive QRS complex, and QS complexes in the anterior-septal leads.^{4,8,9} Another method to identify deviations in the ST-segments and QRS complexes

Presented at the XXII Congress of the European Society of Cardiology, September 2001, Stockholm, Sweden.

This study was supported from unrestricted research grants from Hewlett-Packard (currently Phillips Medical Technologies), Cor Therapeutics (currently Millenium Pharmaceuticals), DuPont Radio Pharmaceuticals (currently Bristol-Myers Squibb Medical Imaging), and EmCare, Inc.

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Figure 1. Calculation of ST-vector magnitude (A) and QRS-vector difference.

in patients with LVH employs the traditional vectorcardiographic (VCG) Frank lead placement for computer generation of a derived 12-lead ECG and X-Y-Z leads from information measured in three-dimensional space.^{10–12} Two measurements that can be made from X-Y-Z leads are the ST-segment vector magnitude (ST-VM), which is traditionally measured at 60 ms after the J-point (Fig. 1A), and the QRS-vector difference (VD) (Fig. 1B). Analysis of ST-VM and QRS-VD trends has been demonstrated to provide important prognostic and diagnostic information in admitted chest pain patients with acute myocardial infarction (AMI) and unstable angina.^{13–26} The ST-VM has also been demonstrated to outperform the initial ECG in identification of AMI in emergency department (ED) chest pain patients without LVH or bundle branch block (BBB) on the initial ECG.²⁷ Currently, no information is available regarding the optimal ST-VM or QRS-VD values predictive of AMI and AO in the ED chest pain patient with LVH on the initial ECG. Theoretically, these optimal values could potentially provide another tool to assist physicians in the initial evaluation and treatment of chest pain patients. The present study investigates the utility of the baseline ST-VM and 2-hour QRS-VD to risk stratify chest pain patients with LVH voltage criteria on the initial 12-lead ECG for AMI and 30-day AO.

METHODS

This prospective study was conducted at a university teaching hospital from January, 1999 to January, 2000, with the approval by the Institutional Review Committee. The study population was derived from 1918 consecutive chest pain patients with suspected ACS who underwent a standardized evaluation protocol that included continuous

VCG monitoring with derived serial 12-lead ECGs, 2-hour delta cardiac serum marker measurements, and selective nuclear stress testing during the initial ED evaluation.^{28,29} Exclusion criteria included patients presenting with chest pain in the presence of a tachyarrhythmia (ventricular tachycardia, supraventricular tachycardia, or rapid atrial fibrillation), patients with pulmonary edema on presentation requiring mechanical ventilation, patients with chest pain not deemed by physician to warrant cardiac workup (obvious nonischemic chest pain and absence of risk factors or preexisting disease), and patients with suspected ACS who did not present with chest pain.

The initial 12-lead ECGs were obtained with the Hewlett-Packard PageWriter XLi Cardiograph (Agilent Technologies). LVH was said to be present if the initial 12-lead ECG met standard voltage criteria for LVH: the greater S-wave voltage in V1 or V2 + the greater R-wave voltage in V5 or V6 > 35 mm; or aVL > 11 mm. Board certified cardiologists blinded to the results of VCG interpreted all ECGs.

Continuous VCG monitoring was performed utilizing the Hewlett-Packard MIDA 1000 (Agilent Technologies, Andover, MA, USA; Ortivus AB, Taby, Sweden), and was continued until either patient was taken for emergent percutaneous coronary intervention (PCI) or until final patient disposition. Three orthogonal VCG leads X, Y, and Z were computed and averaged. In the present study, two VCG parameters were considered: the baseline ST-VM (Fig. 1A) was defined as the VCG sum of the ST-segment deviation 60 ms after the J-point in the X, Y, and Z leads, respectively of the first VCG recorded by the MIDA 1000 utilizing the formula $ST-VM = \sqrt{X^2 + Y^2 + Z^2}$. The 2-hour QRS-VD (Fig. 1B) was defined as the difference between the

area of the 2-hour QRS complex and the baseline complex and measured by the formula $QRS-VD = \sqrt{(\text{area}_X^2 + \text{area}_Y^2 + \text{area}_Z^2)}$. A single trained research assistant blinded to initial ECG interpretation and 30-day outcome, recorded the results of computerized VCG data analysis.

All patients were followed for 30-day outcome after initial ED presentation. Patients with a discharge diagnosis of AMI met World Health Organization diagnostic criteria in effect at the time of the study.^{30,31} AMI was diagnosed if there was ≥ 20 min of chest pain and any one of the following criteria within 24 hours of ED presentation: a serial rise of CK-MB (Abbott Laboratories, Abbott Park, IL) to ≥ 10 ng/ml and CK-MB index $\geq 5\%$; a serial rise in cTnI (Abbott Laboratories, Abbott Park, IL) to ≥ 2 ng/ml; new Q-wave formation in two contiguous leads (based on official ECG interpretation); or death by cardiac or unknown cause. The following cardiac events were registered as endpoints: AMI on presentation, percutaneous transluminal coronary intervention (PCI), coronary artery bypass grafting (CABG), life-threatening complication, or death from cardiac or unknown cause occurring within 30 days of initial ED visit. Life-threatening complications were defined as ventricular fibrillation, sustained ventricular tachycardia, third degree AV block, bradycardic or asystolic arrest, post-ED presentation AMI, cardiogenic shock, or electromechanical dissociation.

Discrete variables are presented by their number and percentages (%). Positive (+) and negative (-) LR were calculated according to the formula $+LR = \text{sensitivity}/(1 - \text{specificity})$ and $-LR = (1 - \text{sensitivity})/\text{specificity}$.³² ROC curves were analyzed utilizing MedCalc[®] 4.2 (MedCalc Software, Belgium).³³ Optimum cut-off values for ST-VM and QRS-VD were defined as the most accurate cut-off value (value with the lowest false-negative and false-positive rate). Sample means were compared with the two-sample unpaired *t* test. Categorical data were compared with chi-square test with the Yates correction for small sample size. Sensitivities and specificities of the paired diagnostic modalities were compared using McNemar's chi-square. Confidence intervals (CI) for difference of proportion were calculated using the formula for paired data.³⁴ A *P* value ≤ 0.05 was considered significant. Data analyses were performed using SYSTAT[®] 10.0 (SPSS, Inc, Chicago, IL, USA).

RESULTS

Over a 13 month time period, a total of 1918 consecutive chest pain patients with suspected AMI underwent a standardized ED evaluation protocol that included continuous VCG monitoring with derived serial 12-lead ECGs, 2-hour delta cardiac serum marker measurements, and selective nuclear stress testing during the initial ED evaluation.^{28,29} Of these patients, a total of 196 patients (10.2%) demonstrated voltage criteria for LVH on the initial ECG. The initial 12-lead ECG was obtained 17 ± 25 min after arrival to the ED. Electrocardiographic injury was identified on the official 12-lead ECG interpretation in 1 (7.1%) of the 14 AMI patients and nondiagnostic in the remaining patients. The baseline ST-VM was obtained 38 ± 34 min after arrival to the ED. Two-hour QRS-VD was recorded in 183 of the study patients (93.4%) including 12 (85.7%) of the AMI patients and 24 (85.7%) of the 30-day AO patients.

Table 1 summarizes 30-day AO in the 196 study patients. Within the 30-day follow-up, 26 (13.3%) of the 196 patients experienced one or more cardiac event. Revascularization was performed in 10 (71.4%) of the 14 AMI patients and 13 (92.9%) of the 14 non-AMI patients. Table 2 summarizes demographic characteristics of patients with 30-day AO versus patients without 30-day AO. There were no statistical differences for any of the demographic characteristics investigated except that patients with 30-day ACS had a higher mean baseline cTnI (4.6 vs 0.44 ng/ml; *P* = 0.0002). Table 3 summarizes mean baseline ST-VM and 2-hour QRS-VD for AMI versus non-AMI patients and for 30-day AO patients versus non-30-day AO patients. Baseline ST-VM was greater in AMI patients versus non-AMI patients (95% CI for difference in means 19.9–94.1) and greater in 30-day AO patients versus non-30-day AO patients (95% CI for difference in means 6.5–61.5). There were no significant

Table 1. Thirty-day Outcome in the 196 Patients with LVH on the Initial ECG

AMI	14 (7.1%)
30-day PTCA/CABG	23 (11.7%)
30-day LT Comp	3 (1.5%)
Death	1 (0.5%)
30-day AO	28 (13.3%)

Table 2. Demographic Characteristics in Patients With and Without 30-day Adverse Outcome

	+30-day AO (N = 28)	–30-day AO (N = 168)	P
Age (yr)	57.4 ± 13.2	54.1 ± 13.4	0.23 [NS]
Mean baseline cTnI (ng/ml)	4.6 ± 14.5	0.44 ± 0.18	0.0002
Male	14 (50%)	85 (50.6%)	1.0 [NS]
Race			
Caucasian	15 (53.6%)	71 (42.3%)	0.12 [NS]
African American	11 (39.3%)	96 (57.1%)	0.12 [NS]
Other	2 (7.1%)	1 (0.6%)	0.07 [NS]
Previous MI	11 (39.3%)	54 (32.1%)	0.60 [NS]
Previous PCI/CABG	8 (28.6%)	39 (23.2%)	0.70 [NS]
Diabetes	9 (32.1%)	37 (22.0%)	0.35 [NS]
Hypertension	25 (89.3%)	143 (85.1%)	0.77 [NS]
Cigarette Use	14 (50%)	77 (45.8%)	0.83 [NS]
Hyperlipidemia	17 (60.7%)	77 (45.8%)	0.21 [NS]
Obesity	10 (35.7%)	75 (44.6%)	0.50 [NS]
Family History	10 (35.7%)	45 (26.8%)	0.46 [NS]

Values are mean ± SD or number of patients in group (%).

differences between 2-hour QRS-VD in AMI patients versus non-AMI patients and in patients with and without 30-day AO. Figures 2A and B represent ROC curves of baseline ST-VM and 2-hour QRS-VD for AMI and 30-day AO, respectively. There were no differences in ROC curve areas between ST-VM and QRS-VD for AMI (0.72 vs 0.62; $P = 0.4$ [NS]) or 30-day AO (0.61 vs 0.59; $P = 0.83$ [NS]). Based on ROC curve analysis, the optimal value for 30-day ACS was $>124 \mu\text{V}$ for the baseline ST-VM and $>21.7 \mu\text{V}$ for 2-hour QRS-VD. Table 4 summarizes sensitivities, specificities, +LR, and –LR of the ST-VM and 2-hour QRS-VD at these optimal values for predicting AMI and 30-day AO. There were no statistically significant differences between baseline ST-VM and 2-hour QRS-VD for AMI (78.6% vs 41.7%; $P = 0.10$ [NS]) or for 30-day AO (60.7%

vs 41.7%; $P = 0.31$ [NS]). QRS-VD was more specific than ST-VM for AMI (78.9% vs 68.7%; $P = 0.017$) and for 30-day AO (80.5% vs 69.6%; $P = 0.009$).

Combining the two tests (i.e., baseline ST-VM $>124 \mu\text{V}$ and/or QRS-VD $>21.7 \mu\text{V}$) had a sensitivity/specificity for AMI of 85.7%/59.3% (+LR 2.1; –LR 0.24) and a sensitivity/specificity for 30-day AO of 71.4%/60.7% (+LR 1.8; –LR 0.47). Patients with either a positive ST-VM or a positive QRS-VD had 8.8 times increased odds of AMI (95% CI 1.9–40.3; $P = 0.003$); 4.3 times increased odds of 30-day PTCA/CABG (95% CI 1.3–13.8; $P = 0.019$); and 3.8 times increased odds of 30-day AO (95% CI 1.6–9.3; $P = 0.003$). The addition of a positive baseline cTnI resulted in no increase in sensitivity for detection of either AMI or 30-day AO.

Table 3. Mean ST-VM and 2-hour QRS-VD for Patients With and Without AMI in the 196 LVH Patients

	+AMI Patients	–AMI	P
Mean ST-VM	165 ± 78 μV	108 ± 67 μV	0.018
Mean QRS-VD	19.5 ± 10.4 μV	16.6 ± 15.6 μV	0.39 [NS]
	+30-day AO	–30-day AO	
Mean ST-VM	141 ± 76 μV	107 ± 67 μV	0.034
Mean QRS-VD	19.7 ± 14.9 μV	16.4 ± 15.4 μV	0.31 [NS]

Values are mean ± SD.

Figure 2. ROC curves of baseline ST-VM and 2-hour QRS-VD for AMI (A) and for 30-day AO (B).

DISCUSSION

Chest pain patients with ECG signs of LVH are a challenge for the evaluating physician due to the difficulties in identifying electrocardiographic injury and ischemia. This study indicates that the simple VCG registration at baseline and 2 hours may help in identifying patients at risk for AMI and adverse cardiac events. Continuous VCG has been demonstrated to provide a reliable noninvasive means to detect reperfusion following fibrinolytic therapy,^{13–15} monitor effects of new antiischemic agents,^{16–18} risk stratify patients with acute coronary syndromes,^{19–26} and for the identification of electrocardiographic injury in ED chest pain patients without LVH or BBB on the initial ECG.²⁷ Currently, there are no prior studies investigating ST-VM or QRS-VD in the general ED chest pain population with LVH on the initial ECG who are undergoing an evaluation for possible ACS.

Lundin et al.¹⁹ studied VCG in 203 patients with AMI (126 patients with Q-wave AMI) during the first 24 hours of hospitalization and found a nonstatistically significant higher median baseline ST-VM (230 vs 180 μ V) and end value QRS-VD (24.1 vs 19.6 μ V) in patients with and without cardiac events. In a related study, Lundin et al.²⁰ investigated ST-

VM and QRS-VD changes in 100 patients admitted with a diagnosis of unstable angina during the first 24 hours of hospitalization and found a statistically significant higher baseline ST-VM (140 vs 80 μ V; $P < 0.01$) and median end value of QRS-VD (10.5 vs 8.8 μ V; $P < 0.05$) in patients with cardiac events versus patients without cardiac events. Abrahamson et al.²⁶ investigated ST-VM during the first 24 hours in 195 patients admitted to the CCU with a diagnosis of unstable angina. Maximum ST-VM over the first 24 hours of hospitalization was found to be an independent predictor of 1-year death (relative risk 1.05 per 10 μ V increase). Utilizing ROC curve analysis, the most accurate ST-VM cut-off in this high-risk population was 144 μ V (sensitivity 59%; specificity 80%; +LR 3; –LR 0.5). Fesmire et al.²⁷ studied VCG in 1722 patients without BBB or LVH on the initial ECG who underwent VCG monitoring during the initial ED evaluation and demonstrated that baseline ST-VM of >121 , >151 , and >175 μ V corresponded to +LR for AMI of 5, 10, and 20, respectively. When combined with physician judgment, these values outperformed subjective ECG interpretation for presence of electrocardiographic injury in AMI patients that suggest this criterion may assist physicians in selecting patients for early reperfusion therapy.

Much attention has been paid in the literature to difficulties of interpreting electrocardiographic injury in the presence of left BBB and paced BBB.^{6,35,36} Little attention has been paid to the difficulties of interpreting injury in the presence of LVH where repolarization abnormalities are frequent and result in significant ST-segment elevation in leads with predominant negative QRS complex and ST-segment depression in leads with predominant positive QRS complex.^{4–6} Our data suggest that electrocardiographic injury is extremely difficult to detect in patients with LVH as only 7% of AMI patients had injury detected on the initial ECG. Utilization of the baseline ST-VM > 124 μ V

Table 4. Sensitivities, Specificities, +LR, and –LR of Baseline ST-VM and 2-hour QRS-VD at the Optimal Value for Predicting AMI and 30-day AO

	Sensitivity	P	Specificity	P	+LR	–LR
AMI						
ST-VM > 124 μ V	78.6 (49.2–95.1)	0.10 [NS]	68.7 (61.4–75.3)	0.017	2.5	0.31
QRS-VD > 21.7 μ V	41.7% (15.3–72.2)		78.9% (72.1–84.8)		2.0	0.74
30-day AO						
ST-VM > 124 μ V	60.7% (40.6–78.5)	0.31 [NS]	69.6% (62.1–76.5)	0.009	2.0	0.56
QRS-VD > 21.7 μ V	41.7% (22.1–63.3)		80.5% (73.5–86.3)		2.1	0.72

increased the sensitivity to 78.6%, but the specificity was only 68.7%. Though our data suggest that both baseline ST-VM and 2-hour QRS-VD supply important prognostic information that may assist physicians in identifying high risk chest pain patients with LVH on the initial ECG, the poor specificity precludes our cut-off values from being utilized as criterion for emergent reperfusion therapy.

The major limitation to this study was the small sample size (only 14 AMIs in 196 LVH patients). However, extrapolating from our data, approximately 14,000 patients with suspected ACS would have to undergo ED VCG monitoring to identify 100 AMI patients. Another limitation of this study was the inability to measure 2-hour QRS-VD in all patients undergoing VCG due to the fact that 13 patients were admitted or taken to the cath lab prior to the 2-hour measurement interval. Other limitations include the short 30-day follow-up time period and the time delay of 20 min from baseline 12-lead ECG registration to VCG registration. This study could also have been improved if we had investigated the maximum and minimum ST-VM during the 2-hour monitoring evaluation period to investigate utility of changes in ST-VM to risk stratify chest pain patients.

The baseline ST-VM and 2-hour QRS-VD risk stratifies ED chest pain patients with voltage criteria for LVH on the initial ECG for AMI and 30-day AO. Multi-institutional studies need to be performed to better investigate utility of ST-VM and QRS-VD to risk stratify chest pain patients with LVH on the initial ECG and to determine if there are any subgroups of LVH patients in whom VCG monitoring can accurately diagnose ACS.

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