Published in final edited form as:

Coron Artery Dis. 2014 March ; 25(2): 138–144. doi:10.1097/MCA.0000000000000062.

Q Wave Area for Stratification of Global Left Ventricular Infarct Size – Comparison to Conventional ECG Assessment using Selvester QRS-score

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Abstract

Objectives—Left ventricular (LV) infarct size is a prognostic determinant after acute myocardial infarction (AMI). ECG data have been used to measure infarct size, but conventional approaches employ multi-parametric algorithms that have limited clinical applicability. This study tested a novel ECG approach – based solely on Q wave area – for calculation of LV infarct size.

Methods—Serial 12-lead ECGs were performed in AMI patients. Computerized software was used to quantify Q wave area (summed across surface ECG leads) and Selvester QRS-score components. ECG analysis was compared to the reference of myocardial infarct size quantified by delayed enhancement cardiac magnetic resonance (DE-CMR).

Results—158 patients underwent ECG during early (4 ± 0.4) and follow-up (29 ± 5 days) post-AMI time points. Selvester QRS-score and Q wave area increased stepwise with LV infarct size ($p < 0.001$). Whereas both methods manifested marked increases at a threshold of 10% LV infarction, magnitude was greater for Q wave area (>2.5 - fold) than Selvester QRS-score (<2 -fold). In ROC analysis, Q wave area (AUC = 0.83-0.86) and Selvester QRS-score (0.82-0.87) manifested similar performance in relation to a 10% infarct cutoff: When Selvester-QRS score and Q wave area thresholds were selected to optimize sensitivity, both methods yielded similar negative predictive value (Q wave area: 89-91% | Selvester QRS-score: 92-94%) although specificity was higher for Q wave area (44-45% vs. 17-25%; $p = 0.01$).

Conclusions—Q wave area provides an index for stratification of LV infarct size that performs similarly to conventional ECG assessment via the Selvester QRS-score for exclusion of large infarction.

Keywords

electrocardiography; acute myocardial infarction; cardiovascular magnetic resonance

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All authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

Conflicts of Interests: None declared

Introduction

Myocardial infarct size is an important prognostic index after acute myocardial infarction (AMI). Electrocardiography (ECG) is widely used as a primary screening test for AMI, for which Q waves comprise a central component of diagnostic criteria.¹ Beyond diagnosis, Q wave morphology has been incorporated into multi-parametric ECG scoring systems that combine Q wave data with other ECG and clinical indices to calculate infarct size^{2, 3}. In comparative studies, the Selvester QRS-score has yielded moderate correlation with infarct size as verified by non-invasive imaging via delayed enhancement cardiac magnetic resonance (DE-CMR).⁴⁻¹³ Use of DE-CMR as a reference standard is supported by multiple studies demonstrating this imaging method to yield excellent agreement with actual infarct size as verified via ex-vivo histopathology.^{14, 15} However, actual magnitude of offset between Selvester score and DE-CMR has been substantial, as evidenced by prior reports of infarct size over- and underestimation by Selvester QRS-score of 27% and 16% respectively.⁵ In addition, the time required for determining the many components of Selvester QRS-score has limited clinical application of this method. Taken together, these findings highlight limitations of current ECG scoring for assessment of infarct size.

Rather than direct infarct size estimation, ECG data can instead be used to discern between patients with large and small infarctions. Such a “threshold-based” approach is consistent with clinical outcomes data, which have shown binary infarct size cutoffs to predict arrhythmic events and mortality.¹⁶⁻¹⁸ Multiple studies have demonstrated both presence and aggregate number of Q waves to increase with infarct size.¹⁹⁻²¹ Our group has recently shown that Q wave area – a parameter that can be measured on routine 12-lead ECG – provides high negative predictive value for exclusion of large (10%) LV infarction.²² Unlike Selvester QRS-score, which incorporates multiple ECG and clinical variables into a multi-parametric formula, Q wave area constitutes a single parameter for assessment of infarct burden, facilitating its application in routine clinical practice.

The aims of the current study were two-fold; (1) to determine whether the Selvester QRS-score manifests threshold based changes in relation to progressive increase in LV infarct size; and, (2) to compare diagnostic performance of Selvester QRS-score to Q wave area for infarct size stratification. The study tested the hypothesis that Q wave area would provide similar stratification of LV infarct size as compared to conventional, multi-parametric ECG assessment via the Selvester score.

Methods

Population

The study population was comprised of patients with first ST elevation AMI enrolled in a prospective registry of post-AMI remodeling (clinical trials # NCT00539045). Subjects were enrolled between September 2006 and March 2013 at Weill Cornell Medical College. Early post-AMI ECGs were acquired during an interval of 3-5 days following AMI. Follow-up ECG and CMR were performed during a pre-specified target of 1 month post-AMI, with both modalities performed on the same day.

Comprehensive demographic data were collected in all patients, including cardiac risk factors and AMI treatment regimen. Coronary angiograms were reviewed for infarct related artery. Informed consent was obtained from each patient in accordance with the Weill Cornell Institutional Review Board (IRB).

Data Acquisition

Cardiac Magnetic Resonance: CMR was performed using 1.5 Tesla scanners (General Electric, Waukesha WI). Exams consisted of two components - (1) cine-CMR for function/morphology, and (2) DE-CMR for infarct quantification.

Cine-CMR was performed using a steady-state free precession sequence (typical parameters: repetition time 3.5 msec, echo time 1.6 msec, flip angle 60°, in-plane spatial resolution 1.9 mm x 1.4 mm). Following cine-CMR, gadolinium was intravenously administered (0.2 mmol/kg). DE-CMR was performed 10–30 minutes thereafter using an inversion recovery pulse sequence. Cine-CMR and DE-CMR images were obtained in matching short and long-axis planes. Short axis images were acquired contiguously through the LV, from the mitral valve annulus through the apex. Long axis images were acquired in standard two-, three- and four-chamber orientations.

Electrocardiography

ECGs were acquired using a standard 12 lead surface electrode recording system (MAC 5000/5500, General Electric, Waukesha WI). Recordings were performed at a sampling rate of 500 Hz. Data was stored digitally using a dedicated (GE Muse) archival system.

Data Analysis

Cardiac Magnetic Resonance: Global LV infarct size (% LV myocardium) was quantified on DE-CMR using the full-width at half-maximum automated method.^{23, 24} LV structure and function, including LVEF and chamber volumes, were quantified using cine-CMR based on planimetry of end-diastolic and end-systolic volumes. LV mass was quantified at end-diastole, with inclusion of papillary muscles and trabeculae within myocardial contours. CMR analysis was performed by a experienced physician (JWW) for whom high intra- and inter-observer reproducibility has previously been reported with regard to quantification of LV infarct size and volumetric indices.^{24, 25}

Electrocardiography

ECGs were quantitatively analyzed using dedicated processing software (Magellan, GE Healthcare, Wauwatosa, WI, USA), which has been previously validated in prior population-based ECG studies.^{26, 27} Multiple aspects of the QRS complex were measured in each surface lead, including duration, maximal amplitude, and geometric area subtended within each respective waveform component. Each Q wave parameter was summed across individual surface leads to calculate total amplitude, duration, and area, which were calculated as unadjusted parameters. Q waves recorded in lead aVR were excluded from all analyses. Established American College of Cardiology/European Society of Cardiology consensus criteria were used to define pathological Q waves (V2-V3: duration 0.02 sec | I, II, aVL, aVF, V4-V6: duration 0.03 sec, amplitude 0.1mV).¹

ECG-based Selvester QRS-scores were calculated according to established, up to date (2011) guidelines.² Scores were based on automated measurements of QRS wave component amplitudes and durations except as noted: Determinations of conduction abnormalities (i.e. left bundle branch, right bundle branch, and left anterior fascicular block), left ventricular hypertrophy, and R wave notching were made by a highly experienced physician (PMO), in accordance with Selvester QRS-score criteria. Aggregate scores were applied to estimate LV infarct size (% myocardium) using a standard formula, for which each Selvester QRS-score point represents 3% LV myocardium.^{3, 5}

Statistical Methods—Comparisons of continuous variables were made using Student's t test (expressed as mean $\pm$ standard deviation) for two group comparisons, and ANOVA for multiple group comparisons. Categorical variables were compared using Chi-square; paired categorical variables were compared using McNemar's test. Pearson correlation coefficients were used to evaluate associations between DE-CMR infarct size and ECG variables. Receiver operating characteristic (ROC) analysis was used to evaluate diagnostic performance of Selvester score and Q wave area for detection of DE-CMR quantified global LV infarct size. Area under the curve (AUC) comparisons were performed based on Z-transformation in accordance with established methods.²⁸ Two-sided $p < 0.05$ was considered indicative of statistical significance. Calculations were performed using SPSS 14.0 (SPSS, Chicago, IL).

Results

The study population comprised 158 post AMI patients who underwent both early post-AMI (4 ± 0.4 days), and follow-up (29 ± 5 days post-AMI) ECG. Baseline clinical characteristics of the population are shown in **Table 1**.

Table 2 compares ECG findings at the early post-AMI and follow-up time points. As shown, aggregate number of pathologic Q waves was slightly higher on early ECG than on follow-up ($p = 0.01$), paralleling changes in Selvester QRS-score ($p < 0.001$). Q wave area and duration were similar at both time points ($p = \text{NS}$) despite minor decreases in Q wave amplitude between early and follow-up ECGs ($p = 0.02$).

Myocardial Infarct Size

Direct ECG Assessment—**Table 3** compares Selvester QRS-score estimated infarct size to the reference of DE-CMR. As shown, the two approaches yielded substantial differences, as evidenced by wide limits of agreement at both early ($-12.7, 23.3\%$ LV myocardium) and follow-up ($-14.4, 21.2\%$) ECG time points. Differences between Selvester QRS-score and DE-CMR were significant for both left anterior descending and right coronary artery infarction ($p < 0.001$).

Threshold-Based ECG Assessment—**Figure 1** stratifies Selvester QRS-score and Q wave area in relation to DE-CMR measured infarction. Both ECG indices increased stepwise with LV infarct size ($p < 0.001$ for trend), with greatest magnitude of difference observed at a threshold of 10% LV infarction. However, as shown, magnitude of increase at 10% infarction was greater for Q wave area than for Selvester QRS-score, as evidenced by over a 2.5-fold difference for Q wave area (2.5-fold early, 2.9-fold follow-up), and a less than 2-fold difference for Selvester QRS-score (1.5-fold early, 1.8-fold follow-up).

Correlations between DE-CMR and ECG demonstrated similar findings in relation to a 10% infarct threshold. Among the overall population, infarct size moderately correlated with Selvester QRS-score on both early ($r = 0.62, p < 0.001$) and follow-up ($r = 0.64; p < 0.001$) ECG; correlations were weaker within groups limited to patients above ($r = 0.33$ - 0.46) and below ($r = 0.18$ - 0.33) a threshold of 10% LV infarction (**Figures 2A-B**). Similarly, Q wave area significantly correlated with infarct size among the overall population ($r = 0.47$ - $0.52; p < 0.001$). However, all correlations between infarct size and Q wave area were weak or non-significant ($r \leq 0.25$) within sub-groups above and below the 10% infarct threshold (**Figures 2C-D**).

Diagnostic performance—**Figure 3** provides receiver operating characteristics curves for Selvester QRS-score and Q wave area in relation to a DE-CMR threshold of 10% LV

infarction. Both ECG indices demonstrated equivalent overall test performance at both the early (Q wave area AUC = 0.83, Selvester QRS-score AUC = 0.82; $p=0.83$) and follow-up (Q wave area AUC = 0.86, Selvester QRS-score AUC = 0.87; $p=0.80$) ECG time-points.

To compare relative utility of each ECG index as a screening test for infarct burden, Selvester QRS-score and Q wave area thresholds were selected to optimize negative predictive value. As shown in **Table 4**, negative predictive value for both methods was similar on early (Selvester QRS-score: 92% | Q wave area: 91%) and follow-up (94% | 89%) ECG. However, Selvester QRS-score yielded lower specificity than did Q wave area at both time points ($p = 0.01$), corresponding to higher false-positive rates (early $n=57$ vs. $n=39$ | follow-up $n=52$ vs. $n=38$). Higher false positive rates for Selvester QRS-score corresponded to lower overall accuracy on early ECG ($p=0.002$), with a similar trend on follow-up ($p=0.07$) compared with Q wave area.

Discussion

This study compared multi-parametric ECG assessment using the Selvester QRS-score to a novel ECG approach – based on Q wave area – for stratification of LV infarct size. There are several key findings: (1) Both Q wave area and Selvester QRS-score increased with LV infarct size ($p<0.001$), with greatest magnitude of difference occurring at a threshold of 10% LV infarct size. (2) Q wave area manifested a more pronounced increase (> 2.5 fold) at a 10% infarct threshold than did Selvester QRS-score (< 2 -fold). (3) When diagnostic thresholds were selected to optimize exclusion of infarcts – 10% myocardium, both ECG methods yielded similar negative predictive value (Selvester QRS-score: 92-94% | Q wave area: 89-91%), although specificity was lower for Selvester QRS-score (17-25%) than for Q wave area (44-45%) at both ECG time points.

While Q wave area provides a new approach to estimate infarct size, multiple prior studies have used Selvester QRS-score for this purpose. The Selvester QRS-score itself has been validated in relation to post-mortem infarct size as measured on histopathology,²⁹⁻³¹ further supporting its use as an ECG reference for comparison. As with our study, prior investigations have compared the Selvester QRS-score to infarct size by DE-CMR, a well-validated non-invasive technique known to closely agree with pathology-evidenced myocardial infarction.^{14, 15} Selvester QRS-score performance in our patient population is in general agreement with prior research findings. For example, our observed correlation between Selvester QRS-score and DE-CMR ($r = 0.62-0.64$) falls within the general range reported among cohorts with acute ($r = 0.33 - 0.79$)^{4-10, 12, 13} and chronic ($r=0.66-0.80$)¹¹ infarcts. Absolute differences between Selvester QRS-score and DE-CMR in our study also paralleled prior published data, which has shown Selvester QRS-score to yield small mean differences ($\Delta = 5.8\%$) but wide limits of agreement ($-15.6, 27.2$) with DE-CMR, suggesting both under- and over-estimation of infarct size by this ECG method.⁵

A central component of our study was to test “threshold” based approach to infarct assessment, whereby the ECG is used to stratify between small and large infarcts, rather than to directly calculate infarct size. This threshold-based approach is predicated on the well-established observation that Q waves (a central component of Selvester QRS-score) can be absent with small infarctions,^{19, 20, 22} and plateau with large infarctions.²² Such an approach is also consistent with recent clinical outcomes studies, which have shown both mortality and arrhythmic risk to be stratified based on binary cutoffs for infarct size.¹⁶⁻¹⁸

Applied clinically, our data suggests that the routine surface ECG may be useful as an initial screening tool to exclude large infarcts ($> 10\%$), thereby avoiding further testing (and associated cost expenditure) in a substantial number of post-AMI patients. Regarding large

infarcts, it is important to recognize that neither Q wave area nor Selvester QRS-score yielded high positive predictive value when thresholds were adjusted to maximize negative predictive value. However, the higher specificity yielded by Q wave area would also be expected to reduce downstream testing (translating into potential cost savings) if the ECG were to be used as an initial means of infarct stratification. For example, results obtained early (4 ± 0.4 days) post MI demonstrated that at nearly equivalent negative predictive value (91% vs. 92%), specificity was >2-fold higher for Q wave area than for Selvester QRS-score (44% vs. 17%), corresponding to a >2-fold increase in the number of subjects ($n=30$ vs. $n=12$) who could avoid unnecessary downstream testing for LV infarct size.

It is important to recognize that for the current study, *both* Q wave area and Selvester QRS-score were calculated using data generated from automated ECG analysis. However, beyond this, substantial differences exist between the methods, which may impact clinical application. For Q wave area, calculations are based on one quantitative parameter – area subtended by the Q wave within each surface ECG lead: No adjustment is made for clinical indices or conditional scoring rules. For Selvester QRS-score, Q wave indices are integrated with multiple other components of the QRS waveform: A total of 35 ECG parameters are measured to test 53 separate scoring criteria. While several are readily quantifiable (e.g. Q and R wave durations/amplitudes), others require analysis of QRS morphology (e.g. “R wave notching”). Selvester score criteria are further modified by clinical indices (e.g. age, gender), atrial and ventricular conduction patterns, as well as left ventricular hypertrophy. A given criterion can become eligible or ineligible for scoring based on which prior criteria have been met within a given ECG lead. The multitude of variables included in this approach, as well as conditional point scoring based on ancillary clinical and/or ECG factors, may explain limited use of Selvester QRS-score routine clinical practice as well as recognition of need to develop automated programs for Selvester QRS-score calculation.³² In contrast, as Q wave area is derived from one parameter, it is reasonable to anticipate that automated measurement could be readily implemented on standard ECG equipment, paralleling widespread use of automation to measure other ECG waveform parameters such as PR and QT interval.

Several limitations should be noted. First, while our study analyzed ECG data from 2 separate post-AMI time points, follow-up did not exceed 4 ± 1 weeks post AMI. However, whereas infarct size is known to decrease in the early post AMI period,^{4, 33} longitudinal follow-up studies have demonstrated infarct stability after 1 month,⁴ supporting the concept that our results are relevant to assessment of patients with chronic infarction. Secondly, the study population was comprised of patients presented following ST elevation AMI. Future studies are warranted to test the utility of this approach in non-ST elevation AMI, as well as non-ischemic cardiomyopathy associated scar.

In conclusion, our finding that Q wave area demonstrated a similar magnitude of agreement with DE-CMR as did Selvester QRS-score supports the notion that the Q wave is the primary determinant of infarct manifestations on the ECG, and that adjustments for additional ECG variables yield minimal benefit in the performance of ECG-based estimation of actual infarct size. Further studies are warranted to test relative utility of ECG based threshold assessment for prognostic risk stratification of post-AMI patients.

Acknowledgments

Sources of Funding: NIH (23 HL102249-01)

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Condensed Abstract

This study tested a novel ECG approach –based on Q wave area (QWAr)– for estimation of left ventricular infarct size. Serial ECGs were performed in 158 post-MI patients (early: 4 ± 0.4 | follow-up: 29 ± 5 days post-MI), and automated software used to quantify QWAr and Selvester QRS-score (SS). ECG analysis was compared to the reference of infarct size by delayed-enhancement cardiac magnetic resonance (DE-CMR). SS and QWAr increased stepwise with infarct size on DE-CMR ($p<0.001$). Whereas both ECG methods manifested marked increases at a 10% infarct threshold, magnitude was greater for QWAr (>2.5 -fold) than SS (<2 -fold). In ROC analysis, QWAr (AUC=0.83-0.86) and SS (0.82-0.87) manifested similar performance in relation to a 10% infarct cutoff. Optimizing thresholds for sensitivity, both methods yielded similar negative predictive value (QWAr: 89-91% | SS: 92-94%) with higher specificity for QWAr (44-45% | 17-25%; $p=0.01$). In conclusion, QWAr provides an index for infarct size stratification that performs similarly to SS for excluding large infarction.

Figure 1. Selvester QRS-score (left) and Q wave area (right) (mean $\pm$ standard deviation), as measured on follow-up ECG, in relation to DE-CMR evidenced infarct size. Note marked increase in both Selvester QRS-score and Q wave area among patients with infarct size 10% LV myocardium (grey bars).

Figure 2.

Scatter plots relating DE-CMR quantified infarct size (y-axis) to ECG quantified Selvester QRS-score (**2A-B**) and Q wave area (**2C-D**) (x-axes). Correlation coefficients and corresponding linear regression lines for each respective scatter plot are shown for subgroups of patients with infarct size $\geq 10\%$ (grey) and $< 10\%$ (black) LV myocardium.

*calculated using infarct cutoff $\geq 10\%$ LV myocardium

Figure 3.

Diagnostic performance of Selvester QRS-score (red) and Q wave area (blue), as measured on early (left) and follow-up (right) ECG in relation to a cutoff of infarct size $\geq 10\%$ LV myocardium.

Table 1**Population Characteristics**

Parameter	
DEMOGRAPHICS	
Age (year)	56 ± 12
Male gender	84% (132)
Atherosclerosis Risk Factors	
Hypertension	41% (64)
Diabetes mellitus	22% (35)
Hypercholesterolemia	48% (76)
Tobacco use	31% (49)
Family history	26% (41)
STEMI PROFILE	
Revascularization Strategy	
Primary PCI (%)	75% (118)
Primary thrombolysis (%)	25% (40)
Infarct Related Artery	
Left anterior descending	55% (87)
Left circumflex	9% (14)
Right coronary	36% (57)
IMAGING CHARACTERISTICS	
Left Ventricular Structure and Function	
LV ejection fraction (%)	54 ± 11
End-diastolic volume (ml/m ²)	76 ± 16
End-systolic volume (ml/m ²)	36 ± 15
Myocardial mass (gm/m ²)	65 ± 13
Infarct Size	
LV hyperenhancement (% myocardium)	12 ± 9

Table 2

ECG Findings

Parameter	Early post-AMI	Follow-up	P
ECG PARAMETERS			
Intervals			
RR interval (msec)	854 ± 147	1020 ± 165	<0.001
QRS duration (msec)	92 ± 13	91 ± 12	0.64
QTc duration (msec)	434 ± 30	424 ± 28	<0.001
QRS axis (°)	19 ± 48	20 ± 46	0.78
Q wave Indices			
Pathologic Q waves	73%	70%	0.36
Number of Pathologic Q waves	1.5 ± 1.4	1.3 ± 1.2	0.01
Q wave area (mV·msec)	42 ± 41	40 ± 45	0.30
Q wave duration (msec)	205 ± 108	194 ± 115	0.11
Q wave amplitude (mV)	1.9 ± 1.5	1.7 ± 1.6	0.02
Selvester QRS-score (% LV myocardium)	17.7 ± 11.3	15.8 ± 11.4	<0.001
Conduction Abnormalities			
Right Bundle Branch Block	2% (3)	1% (2)	1.00
Left Bundle Branch Block	-	-	-
Left Anterior Fascicular Block	3% (5)	1% (2)	0.25
Bifascicular Block [*]	1% (1)	1% (2)	1.00
Left Ventricular Hypertrophy	4% (6)	1% (1)	0.06

Boldface indicates p values < 0.05

^{*} right bundle and left anterior fascicular block

Table 3

Infarct size by DE-CMR and Selvester QRS-score

LV infarct size (% LV myocardium)	DE-CMR	Selvester QRS-score					
		Early	Δ	P	r	P	r
Overall study population	12.4 $\pm$ 8.8	17.7 $\pm$ 11.3	5.3 $\pm$ 9.0	<0.001	0.62	<0.001	0.64
Infarct related artery							
Left Anterior Descending	14.8 $\pm$ 8.7	21.6 $\pm$ 11.1	6.8 $\pm$ 8.7	<0.001	0.64	<0.001	0.61
Left Circumflex	10.0 $\pm$ 7.9	11.8 $\pm$ 9.4	1.7 $\pm$ 8.0	0.43	0.59	0.37	0.71
Right Coronary Artery	9.3 $\pm$ 8.1	13.2 $\pm$ 9.8	3.8 $\pm$ 9.4	0.003	0.46	0.01	0.58

Diagnostic Performance of ECG-based Infarct Size Assessment for 10% Left Ventricular Infarct*

Table 4

A. Early Post-AMI						
	Sensitivity	Specificity	Accuracy	Positive Predictive Value	Negative Predictive Value	
Selvester QRS-score 1	99%	17%	63%	61%	92%	
Q wave Area 8.68 mV•msec	97%	44% [†]	73% [‡]	69%	91%	
B. Follow-up						
	Sensitivity	Specificity	Accuracy	Positive Predictive Value	Negative Predictive Value	
Selvester QRS-score 1	99%	25%	66%	63%	94%	
Q wave Area 4.35 mV•msec	96%	45% [†]	73% [‡]	69%	89%	

*cutoffs optimized for sensitivity/negative predictive value

* cutoffs optimized for sensitivity/negative predictive value

[†] p<0.001 (vs, Selvester QRS-score)

[‡] p=0.002 (vs, Selvester QRS-score)

[†] p=0.01 (vs, Selvester QRS-score)

[‡] p=0.07 (vs, Selvester QRS-score)