Published in final edited form as:

Am J Cardiol. 2013 July 15; 112(2): . doi:10.1016/j.amjcard.2013.03.013.

Usefulness of Q-Wave Area for Threshold-Based Stratification of Global Left Ventricular Myocardial Infarct Size

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Abstract

Left ventricular (LV) infarct size affects prognosis after acute myocardial infarction (AMI). Delayed enhancement cardiac magnetic resonance (DE-CMR) provides accurate infarct quantification but is unavailable or contraindicated in many patients. This study tested whether simple electrocardiography (ECG) parameters can stratify LV infarct size. One hundred fifty-two patients with AMI underwent DE-CMR and serial 12-lead ECG. Electrocardiograms were quantitatively analyzed for multiple aspects of Q-wave morphology, including duration, amplitude, and geometric area (QWAr) summed across all leads except aVR. Patients with pathologic Q waves had larger infarcts measured by DE-CMR or enzymes (both $p < 0.001$), even after controlling for infarct distribution by CMR or x-ray angiography. Comparison between early (4 ± 0.4 days after AMI) and follow-up (29 ± 6 days) ECG demonstrated temporal reductions in Q-wave amplitude (1.8 ± 1.4 vs 1.6 ± 1.6 mV; $p = 0.03$) but not QWAr (41 ± 38 vs 39 ± 43 mV•ms; $p = 0.29$). At both times, QWAr augmented stepwise with DE-CMR quantified infarct size ($p < 0.001$). QWAr increased markedly at 10% LV infarct threshold, with differences more than threefold on early ECG (59 ± 39 vs 18 ± 20 mV•ms; $p < 0.001$) and nearly fivefold (59 ± 46 vs 13 ± 16 mV•ms; $p < 0.001$) on follow-up. Diagnostic performance compared with a 10% infarction cutoff was good on early (area under the curve = 0.84) and follow-up (area under the curve = 0.87) ECG. Optimization of sensitivity (95% to 98%) enabled QWAr to exclude affected patients with 90% to 94% negative predictive value at each time point. In conclusion, LV infarct size is accompanied by stepwise increments in Q-wave morphology, with QWAr increased three- to fivefold at a threshold of 10% LV infarction. Stratification based on QWAr provides excellent negative predictive value for exclusion of large (10%) LV infarct burden.

Left ventricular (LV) infarct size is an established determinant of adverse clinical outcomes after acute myocardial infarction (AMI).^{1–4} Myocyte necrosis provides a proarrhythmic substrate and has been shown to predict risk for ventricular tachyarrhythmias independent of contractile dysfunction.^{3,5} Accordingly, stratification of LV infarct size is of prognostic importance for post-AMI patients. Delayed enhancement cardiac magnetic resonance (DE-CMR) is well validated as a noninvasive reference standard for LV infarct quantification.^{6,7} DE-CMR has been used clinically to measure infarct size and transmural. However, population-based use of DE-CMR as a primary means of stratifying infarct burden would

entail substantial costs and be infeasible in many patients. Electrocardiography (ECG), a routine component of post-AMI care, is inexpensive and readily obtainable in nearly all patients. Although it is known that small infarcts can occur without pathologic Q waves on electrocardiogram,^{8–10} it is possible that a size threshold exists above which Q-wave morphology can be used to stratify infarct burden. ECG might therefore be useful as a primary screening test to exclude infarcts below a given threshold, with more sophisticated testing reserved to quantify infarct size in patients with specific Q-wave abnormalities in the QRS complex. To date, studies comparing DE-CMR with ECG-based infarct quantification have used scoring systems that encompass several aspects of the QRS waveform,^{11–14} without addressing whether Q-wave morphology alone can serve as a marker of global infarct burden. The aims of this study were twofold: (1) to evaluate whether Q-wave morphology, as assessed on early and follow-up post-AMI ECG, parallels global infarct size on DE-CMR, and (2) to determine whether a quantitative cutoff exists whereby Q-wave indexes can be used to stratify patients above and below a given threshold for global LV infarct size.

Methods

The population comprised patients with first ST elevation AMI enrolled in a prospective registry of post-AMI remodeling (ClinicalTrials.gov no. NCT00539045) from September 2006 to October 2012. In accordance with established criteria, AMI was defined by chest pain with ST segment elevation in ≥2 contiguous ECG leads and elevated cardiac enzymes (more than ninety-ninth percentile).¹⁵ Patients were excluded for previous AMI or contraindication to CMR. Comprehensive clinical data were collected, including cardiac risk factors, coronary artery disease history, and treatment regimen. Coronary angiograms were reviewed for infarct related artery and lesion-based (Alberta Provincial Project for Outcome Assessment in Coronary Heart Disease [APPROACH]) myocardial jeopardy score.¹⁶ Electrocardiograms were acquired at 2 time points (1) <1 week (target 3 to 5 days) after AMI, and (2) 2 to 6 weeks after AMI. In all patients, CMR was performed on the same day as follow-up (2- to 6-week) ECG. The study was conducted in accordance with the Weill Cornell Institutional Review Board.

Electrocardiograms were acquired using a standard 12-lead surface electrode recording system (MAC 5000/550, General Electric, Waukesha, Wisconsin). Recordings were performed at a sampling rate of 500 Hz. Data were stored digitally using a dedicated (GE Muse, General Electric) archival system. Electrocardiograms were quantitatively analyzed using a dedicated processing software (Magellan, GE Healthcare, Wauwatosa, Wisconsin). A band-pass filter of 0.05 to 150 Hz was applied before measurement, consistent with American Heart Association/American College of Cardiology recommendations.¹⁷ Multiple aspects of Q-wave morphology were measured in each surface lead, including duration, maximal amplitude, and geometric area subtended within each respective Q wave. Q waves were defined as the first negative deflection of the QRS complex. If a QS complex pattern was seen, the entire QS wave was defined as a Q wave. To minimize the impact of ECG noise artifact, Q-wave area (QWAr) was only recorded when $\geq 160 \mu\text{V}\cdot\text{ms}$. Respective Q-wave parameters (amplitude, duration, and area) were similarly measured for all other QRS waveform deflections as recorded within all ECG leads.

Each Q-wave parameter was summed across individual surface leads to calculate total amplitude, duration, and area, which were calculated as unadjusted parameters and with adjustment for absolute QRS data (i.e., total QWAr/total QRS area). Q waves as recorded in lead aVR were excluded from all analyses. Established consensus criteria were used to define binary presence or absence of pathologic Q waves (V_2 to V_3 : duration ≥ 0.02 second | I, II, aVL, aVF, V_4 to V_6 : duration ≥ 0.03 second, amplitude ≥ 0.1 mV).¹⁵

CMR was performed using 1.5-Tesla scanners (General Electric, Waukesha, Wisconsin). Exams consisted of 2 components: (1) cine-CMR for function or 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 milliseconds, echo time 1.6 milliseconds, flip angle 60°, inplane spatial resolution 1.9×1.4 mm). After cine-CMR, gadolinium was intravenously administered (0.2 mmol/kg) and DE-CMR performed 10 to 30 minutes thereafter using a segmented inversion recovery sequence. Cine-CMR and DE-CMR images were obtained in matching short- and long-axis planes. Short-axis images were acquired throughout the LV (6-mm slice thickness, 4-mm gap) from the level of the mitral valve annulus through the apex. Long-axis images were acquired in standard 2-, 3-, and 4-chamber orientations.

Global LV infarct size (%LV myocardium) was quantified on DE-CMR using the full width at half maximum method, for which high reproducibility has been reported.^{18,19} Regional infarct location and transmural extent was scored using an established 17-segment model or 5-point-per-segment scoring system (0 = no hyper-enhancement; 1 = 1% to 25%; 2 = 26% to 50%; 3 = 51% to 75%; 4 = 76% to 100%).²⁰ LV structure and function were quantified using cine-CMR. LV mass was quantified at end-diastole, with inclusion of papillary muscles and trabeculae within myocardial contours. LV ejection fraction and cavity volumes were based on end-diastolic and end-systolic endocardial contours of contiguous short-axis images. Regional LV wall motion was scored using a 17-segment model. Segmental function was graded as 0 = normal contraction, 1 = mild hypokinesia, 2 = moderate hypokinesia, 3 = severe hypokinesia, 4 = akinesia, and 5 = dyskinesia.

Comparisons of continuous variables were made using Student's *t* test (expressed as mean $\pm$ standard deviation) for 2 group comparisons and analysis of variance for multiple group comparisons. Categorical variables were compared using chi-square or Fisher's exact tests when <5 outcomes were expected per cell; paired categorical variables were compared using McNemar's test. Linear regression was used to evaluate multivariable associations between Q-wave parameters and DE-CMR. Receiver operating characteristic analysis was used to evaluate diagnostic performance of Q-wave parameters 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.²¹ Two-sided $p < 0.05$ was considered indicative of statistical significance. Calculations were performed using SPSS 14.0 (SPSS, Chicago, Illinois).

Results

The population comprised 152 patients who had both early (4 ± 0.4 days) and follow-up (29 ± 6 days post-AMI) ECG performed after AMI. In all patients, follow-up ECG was performed on the same day as CMR.

Table 1 details baseline characteristics of the study population, with stratification based on presence or absence of follow-up ECG-evidenced pathologic Q waves (defined based on established criteria).¹⁵ As expected, affected patients had larger global infarct burden, whether measured by cardiac enzymes or DE-CMR evidenced hyperenhancement (both $p < 0.001$). Consistent with this, pathologic Q waves were associated with hemodynamic instability as assessed based on need for pharmacologic and/or mechanical support ($p = 0.04$).

Regarding infarct distribution, pathologic Q waves were more common with anterior wall infarction, whether assessed directly by DE-CMR or angiographically based on left anterior descending culprit vessel involvement (both $p = 0.001$). In multivariate analysis, pathologic

Q waves were independently associated with DE-CMR quantified global infarct size (odds ratio [OR] = 1.3 per %LV myocardium, 95% confidence interval [CI] = 1.2 to 1.4; $p < 0.001$) after controlling for anterior wall involvement (OR = 1.5, 95% CI = 0.6 to 4.0; $p = 0.38$) (model chi-square = 65.6; $p < 0.001$). Conventional assessment of infarct size and distribution demonstrated a similar relation, as evidenced by an independent association between Q waves and peak serum creatinine phosphokinase (OR = 1.1 per 100 u/L, 95% CI = 1.1 to 1.2; $p < 0.001$) after controlling for left anterior descending infarction on angiography (OR = 1.7, 95% CI = 0.7 to 4.3; $p = 0.23$; model chisquare = 52.4; $p < 0.001$).

Prevalence of pathologic Q waves, as scored based on presence or absence in any ECG lead, was similar between time points (72% vs 68%, $p = 0.36$). However, aggregate number of Q waves was slightly higher on early post-AMI ECG (1.4 ± 1.4) compared with follow-up (1.2 ± 1.2 ; $p < 0.01$). Figure 1 compares Q-wave morphology between early and follow-up ECG. Consistent with decrements in absolute number of pathologic Q waves, total Q-wave amplitude (QWAm; summed across ECG leads) was lower at follow-up (1.6 ± 1.6 vs 1.8 ± 1.4 mV; $p = 0.03$). However, there was only a trend toward lower total Q-wave duration (QWDur; 190 ± 109 vs 202 ± 105 ; $p = 0.06$), and total QWAr was similar between time points (39 ± 43 vs 41 ± 38 mV•ms; $p = 0.29$).

As shown in Table 2, QWAr, an aggregate parameter accounting for both duration and amplitude, correlated with global infarct size on early and follow-up ECG. Similarly, global LV infarct size correlated with increases in both total QWAm and QWDur. However, multivariate analysis demonstrated differences between time points: Early post-AMI, total QWAm independently correlated with infarct size, whereas total QWDur did not. Conversely, QWDur on follow-up ECG independently correlated with infarct size, with nonsignificance for QWAm.

QWAr increased stepwise in relation to global LV infarct size, whether quantified based on DE-CMR (Figure 2) or measured indirectly based on cardiac enzymes. Results were similar at each ECG time point (all $p < 0.001$ for trend). Of note, QWAr increased markedly when patients were stratified using a DE-CMR infarct threshold of 10% LV myocardium, as evidenced by more than a threefold difference on early ECG (59 ± 39 vs 18 ± 20 mV•ms, $p < 0.001$) and nearly a fivefold difference (59 ± 46 vs 13 ± 16 mV•ms, $p < 0.001$) on follow-up. Stratification based on enzymatic infarct size demonstrated QWAr to similarly discriminate patients in the lowest creatinine phosphokinase quartile from the remainder of the population, with respective differences of threefold (50 ± 39 vs 17 ± 20 mV•ms) and 4.5-fold (49 ± 45 vs 11 ± 15 mV•ms) on early and late ECG (both $p < 0.001$).

Figure 3 provides receiver operating characteristics curves for QWAr in relation to DE-CMR evidenced infarct size $\geq 10\%$ LV myocardium. QWAr on both early (AUC = 0.84) and follow-up (AUC = 0.87). ECG demonstrated good diagnostic performance, with nonsignificant differences between time points ($p = 0.49$). As shown in Table 3, optimization of diagnostic sensitivity (95% to 98%) allowed QWAr to exclude the presence of $\geq 10\%$ LV infarction in the overwhelming majority of patients, as evidenced by 90% negative predictive value at each ECG time point.

Discussion

This study provides new insights concerning the relation between global LV infarct burden and Q-wave morphology. Results demonstrate that LV infarct size is accompanied by stepwise increments in Q-wave morphology, with a marked (three- to fivefold) increase in QWAr at a threshold of 10% LV myocardium ($p < 0.001$). QWAr quantified on both early and follow-up ECG demonstrated similar discriminatory capability for 10% global LV

infarction (AUC = 0.84 to 0.87) and excellent negative predictive value (90% to 94%), despite time-dependent temporal changes in individual components of QWDur and QWAmp.

To the best of our knowledge, all previous studies that have compared DE-CMR and ECG data have either categorized Q waves in a binary fashion or incorporated Q-wave data into multifaceted scoring systems.^{8–14} Consistent with our findings, data in smaller cohorts have demonstrated patients with pathologic Q waves to have larger infarcts on DE-CMR.^{8–10} However, although these studies measured infarct size on DE-CMR as a continuous variable, Q waves were categorized as present or absent, prohibiting evaluation of linear relations between infarct burden and Q waves. Our study used a different approach in which both ECG and DE-CMR were independently analyzed using quantitative methods, enabling variables from both methods to be measured in a continuous fashion.

Concerning ECG manifestations of LV infarct size, our data highlight the importance of time-dependent changes in Q-wave morphology. Although infarct size independently correlated with total QWAmp (partial $r = 0.28$, $p = 0.001$) measured early post-AMI, follow-up ECG demonstrated an independent correlation with QWDur (partial $r = 0.30$, $p < 0.001$). Time-dependent differences in ECG or DE-CMR multivariate models were paralleled by a significant reduction in QWAmp between early and follow-up ECG. Although exact mechanisms are not established, it is likely that dynamic changes in Q-wave morphology relate to post-AMI remodeling, with resultant alterations in electroanatomical conduction. Infarct shrinkage is known to occur after AMI, as evidenced by studies reporting 14% to 37% reductions in hyperenhancement size between early and follow-up DE-CMR.^{12,22,23} It is possible that infarct shrinkage may reduce QWAmp, with lesser reductions in QWDur resulting in nonsignificant changes in QWAr ($\Delta = 2.4 \pm 28 \text{ mV}\cdot\text{ms}$, $p = 0.29$) as observed in our study. Regardless of mechanism, our data demonstrate DE-CMR quantified infarct size to manifest near equivalent correlations between QWAr as measured on early ($r = 0.49$ to 0.52 , $p < 0.001$) and follow-up ($r = 0.44$ to 0.51 , $p < 0.001$) ECG, supporting use of this integrated geometric parameter for study of global infarct burden.

The moderate correlation between DE-CMR and QWAr observed in our population is consistent with previous studies that have used ECG scoring systems to estimate infarct size.^{11–14} However, although such scoring systems incorporate both QWAmp and QWDur, they also encompass multiple other aspects of the ECG waveform, yielding uncertainty as to whether the Q wave alone can be used as a marker of infarct burden. Additionally, previous studies have reported variable magnitude of correlation between DE-CMR and ECG infarct score ($r = 0.33$ to 0.79).^{11–14} Our data suggest that one reason for difference in agreement between ECG and DE-CMR may relate to infarct size: QWAr differed three- to fivefold among patients stratified using a threshold of 10% LV myocardium and demonstrated lesser magnitude of difference among patients further stratified above or below this cutoff.

Our finding of a threshold effect of 10% LV infarction on QWAr bears important implications for clinical risk stratification. Previous outcomes studies have demonstrated DE-CMR quantified infarct size to stratify arrhythmic risk, with a threshold based approach shown to stratify risk independent of LV ejection fraction.³ Consistent with this concept, a 10% infarct threshold has been used in an active multicenter study (Defibrillators to Reduce Risk by Magnetic Resonance Imaging Evaluation—DETERMINE ICD), designed to test whether DE-CMR can be used to stratify need for implantable cardioverter defibrillator.²⁴ Although CMR is established as a precise tool for infarct quantification,^{6,7} broad clinical application would bear substantial economic costs and would be contraindicated in a substantial number of patients. Our results demonstrate that available data from 12-lead surface ECG, routinely obtained after AMI at a fraction of the cost of CMR, can be used to

exclude a large majority of patients with high (> 10% infarct) burden. Thus, it is plausible that an integrated strategy of ECG screening (using a high sensitivity/negative predictive value threshold for QWAr) could be useful as a first-line screen for stratification of infarct burden. DE-CMR could then be reserved for at-risk patients with large QWAr, thereby optimizing diagnostic strengths of each method while minimizing cost. It should be noted that applicability of this approach is limited by the requirement of ECG-processing software that is not yet in widespread clinical use. However, established methods for ECG-based infarct size assessment are similarly dependent on the use of ECG-processing software for practical application.^{25,26} Future studies are warranted to test the prognostic utility of integrated ECG-based risk stratification of LV infarct burden for arrhythmogenic risk assessment.

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Figure 1. Q-wave evolution. QWAmp, QWDur, and QWAr as measured on early ECG (*black bars*) compared with follow-up ECG (*gray bars*). Data presented as mean $\pm$ SD.

Figure 2.

QWAr in relation to global LV infarct size. (A) QWAr (mean $\pm$ SD), as measured on early ECG (*top*) and follow-up ECG (*bottom*), in relation to DE-CMR-evidenced infarct size. Note marked increase in QWAr among patients with infarct size $\geq 10\%$ LV myocardium (*black bars*). (B) QWAr in relation to peak creatinine phosphokinase. Note stepwise increase in QWAr across population-based enzymatic quartiles, paralleling DE-CMR findings.

Figure 3.

Receiver operator characteristics analysis. Diagnostic performance of QWAr, as measured on early (*blue*) and follow-up (*red*) ECG, for detection of infarct size $\geq 10\%$ LV myocardium. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 1

Population characteristics*

Parameter	Overall (n = 152)	Q-Wave + (n = 104)	Q-Wave – (n = 48)	p Value
Age (yr)	56 ± 12	55 ± 12	58 ± 13	0.24
Men	126 (83%)	91 (88%)	35 (73%)	0.03
Hypertension	59 (39%)	38 (37%)	21 (44%)	0.40
Diabetes mellitus	34 (22%)	23 (22%)	11 (23%)	0.91
Hypercholesterolemia	72 (47%)	45 (43%)	27 (56%)	0.14
Tobacco use	46 (30%)	25 (24%)	21 (44%)	0.01
Family history of coronary disease	39 (26%)	28 (27%)	11 (23%)	0.60
Previous coronary revascularization	8 (5%)	4 (4%)	4 (8%)	0.26
Revascularization strategy				
1° PCI	115 (76%)	78 (75%)	37 (77%)	0.78
Primary thrombolysis	37 (24%)	26 (25%)	11 (23%)	0.78
Thrombolysis with emergent PCI	22 (15%)	17 (16%)	5 (10%)	0.33
Pharmacologic/mechanical support	31 (20%)	26 (25%)	5 (10%)	0.04
Cardiovascular medications				
β blocker	144 (95%)	101 (97%)	43 (90%)	0.05
ACE inhibitor/angiotensin receptor blocker	91 (60%)	66 (64%)	25 (52%)	0.18
HMG-CoA reductase inhibitor	146 (96%)	99 (95%)	47 (98%)	0.67
Thienopyridine	147 (97%)	101 (97%)	46 (96%)	0.65
Aspirin	152 (100%)	104 (100%)	48 (100%)	-
IIb/IIIa antagonists	122 (82%)	89 (86%)	33 (72%)	0.03
Infarct size and distribution				
DE-CMR				
LV hyperenhancement (g)	15 ± 12	19 ± 12	6 ± 6	<0.001
Hyperenhancement (% myocardium)	12 ± 9	16 ± 8	5 ± 4	<0.001
Anterior wall involvement	80 (53%)	67 (64%)	13 (27%)	<0.001
Lateral wall involvement	51 (34%)	34 (33%)	17 (35%)	0.74
Inferior wall involvement	86 (57%)	56 (54%)	30 (63%)	0.32
Angiography				
Jeopardy score	26 ± 11	29 ± 10	21 ± 11	<0.001
Infarct-related artery				
Left anterior descending	84 (55%)	67 (64%)	17 (35%)	0.001
Left circumflex	14 (9%)	4 (4%)	10 (21%)	0.002
Right coronary	54 (36%)	33 (32%)	21 (44%)	0.15
Cardiovascular enzymes				
Creatine phosphokinase [†]	2,284 ± 1,964	2,906 ± 2,043	968 ± 811	<0.001
LV function/geometry				
Ejection fraction (%)	54 ± 11	51 ± 10	62 ± 8	<0.001
Wall motion score	0.7 ± 0.5	0.9 ± 0.5	0.3 ± 0.3	<0.001
End-diastolic volume (ml/m ²)	76 ± 16	78 ± 16	69 ± 13	0.001

Parameter	Overall (n = 152)	Q-Wave + (n = 104)	Q-Wave – (n = 48)	p Value
End-systolic volume (ml/m ²)	36 ± 15	40 ± 15	27 ± 10	<0.001
Myocardial mass (g/m ²)	65 ± 13	67 ± 13	61 ± 11	0.003

Boldface indicates p values <0.05.

ACE = angiotensin converting enzyme; HMG-CoA = 3-hydroxy-3-methylglutaryl-coenzyme A; PCI = percutaneous coronary intervention.

* Stratified based on consensus criteria for pathological QWDur (≥ 20 ms V2 to V3, ≥ 30 ms I, II, aVL, aVF, V4 to V6) and QWAmp (≥ 0.1 mV I, II, aVL, aVF, V4 to V6).¹⁶

[†] Data available for 88% of population (n = 134).

Table 2

Q-wave-based correlations with DE-CMR quantified infarct size

Early After AMI					
Q-wave Indices	Correlation (r)	p Value	Multivariate		
QWAr	0.49	<0.001			
Adjusted QWAr	0.52	<0.001			
Model r = 0.52					
			Partial r	β	p
QWDur	0.46	<0.001	0.11	0.15	0.17
QWAmp	0.51	<0.001	0.28	0.39	0.001
Model r = 0.56					
			Partial r	β	p
Adjusted QWDur (Qw-dur/QRS-dur)	0.43	<0.001	−0.07	−0.10	0.42
Adjusted QWAmp (Qw-amp/QRS-amp)	0.55	<0.001	0.39	0.64	<0.001
Follow-Up After AMI					
Q-wave indices	Correlation (r)	p	Multivariate		
QWAr	0.44	<0.001			
Adjusted QWAr	0.51	<0.001			
Model r = 0.52					
			Partial r	β	p
QWDur	0.51	<0.001	0.30	0.45	<0.001
QWAmp	0.44	<0.001	0.06	0.08	0.49
Model r = 0.51					
			Partial r	β	p
Adjusted QWDur (Qw-dur/QRS-dur)	0.50	<0.001	0.16	0.28	0.049
Adjusted QWAmp (Qw-amp/QRS-amp)	0.49	<0.001	0.15	0.25	0.08

Table 3

Diagnostic performance of QWAr for 10% LV infarct

	Sensitivity	Specificity	Accuracy	Positive Predictive Value	Negative Predictive Value
Early after AMI					
QWAr	98%	45%	74%	69%	94%
>8.660 mV•msec *					
Follow-up					
QWAr	95%	52%	76%	72%	90%
<5.385 mV•msec *					

* Cutoff optimized for sensitivity/negative predictive value.