Pathological Q Waves in Myocardial Infarction in Patients Treated by Primary PCI

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OBJECTIVES In the present study, we investigated the association of pathological Q waves with infarct size. Furthermore, we investigated whether Q-wave regression was associated with improvement of left ventricular ejection fraction (LVEF), infarct size, and left ventricular dimensions in ST-segment elevation myocardial infarction (STEMI) patients with early Q-wave formation compared with patients without or persistent pathological Q waves.

BACKGROUND The criteria for pathological Q waves after acute myocardial infarction (MI) have changed over the years. Also, there are limited data regarding correlation of Q-wave regression and preservation of LVEF in patients with an initial Q-wave MI.

METHODS Standard 12-lead electrocardiograms (ECGs) were recorded in 184 STEMI patients treated with primary percutaneous coronary intervention (PCI). ECGs were recorded before and following PCI, as well as at 1, 4, 12, and 24 months of follow-up. An ECG was scored as Q-wave MI when it showed Q waves in 2 or more contiguous leads according to the 4 readily available clinical definitions used over the years: "classic" criteria, Thrombolysis In Myocardial Infarction criteria, and 2000 and 2007 consensus criteria. Cardiac magnetic resonance (CMR) examination was performed at 4 ± 2 days after reperfusion and repeated after 4 and 24 months. Contrast-enhanced CMR was performed at baseline and 4 months.

RESULTS The classic ECG criteria showed strongest correlation with infarct size as measured by CMR. The incidence of Q-wave MI according to the classic criteria was 23% 1 h after PCI. At 24 months of follow-up, 40% of patients with initial Q-wave MI displayed Q-wave regression. Patients with a Q-wave MI had larger infarct size and lower LVEF on baseline CMR (24 ± 10% LV mass and 37 ± 8%, respectively) compared with patients with non–Q-wave MI (17 ± 9% LV mass, p < 0.01, and 45 ± 8%, p < 0.001, respectively). Patients with Q-wave regression displayed significantly larger LVEF improvement in 24 months (9 ± 11%) as compared with both persistent Q-wave MI (2 ± 8%) as well as non–Q-wave MI (3 ± 8%, p = 0.04 for both comparisons).

CONCLUSIONS Association of Q waves with infarct size is strongest when using the classic Q-wave criteria. Q-wave regression is associated with the largest improvement of LVEF as assessed with CMR.   (J Am Coll Cardiol Img 2013;6:324–31) © 2013 by the American College of Cardiology Foundation

The electrocardiogram (ECG) plays a pivotal role in the diagnosis of myocardial infarction (MI), due to low costs and universal availability. On the basis of the ECG, patients presenting with an acute coronary syndrome are stratified in the acute setting into ST-segment elevation myocardial infarction (STEMI) or non–ST-segment elevation myocardial infarction. After the acute setting, the ECG may show pathological Q

waves. Pathological Q waves are considered the classic ECG sign of necrosis and represent the area of myocardium that cannot be depolarized. The emergence of Q waves on the ECG constitutes important prognostic value for clinicians. Several post-mortem (1) and cardiac magnetic resonance (CMR) studies (2–4) showed that Q-wave MIs are larger.

A variety of definitions for pathological Q waves have been published over the years. Earlier studies defined the pathological Q-wave as being more than 0.04 s of duration and with an amplitude of more than 25% of the corresponding R-wave (5–7), a criterion used since 1934. In 1999, the TIMI (Thrombolysis In Myocardial Infarction) investigators classified Q waves as being pathological if they lasted for more than 0.03 s based on the Selvester criteria (8). Two consensus documents in 2000 (9) and 2007 (10) redefined the ECG criteria yet again (Table 1).

Moreover, it has been shown that in the era of thrombolytic therapy, Q waves can disappear partially or completely during the evolution of myocardial infarction (11–13). This was observed in approximately 15% of the patients with a STEMI. It is not clear though whether such Q-wave regression can be used as a clinical tool because it is related to shrinkage in infarct size or improvement of left ventricular ejection fraction (LVEF).

We hypothesize that the classic, more strict, criteria of Q-wave MI correlate best with infarct size. Moreover, we hypothesize that Q-wave regression is related to shrinkage in infarct size and improvement of LVEF. Therefore, in the present study, we investigated the association of pathological Q waves with infarct size, focusing on readily available clinical definitions of pathological Q-wave formation. Furthermore, we investigated whether Q-wave regression was associated with improvement of LVEF, infarct size, and left ventricular dimensions in STEMI patients with early Q-wave formation compared with patients without or persistent pathological Q waves.

METHODS

Patient population. The study population consisted of 200 STEMI patients treated by primary percutaneous coronary intervention (PCI) undergoing CMR for research indications in 7 Dutch primary PCI centers. Patients with a history of myocardial infarction were excluded. All patients had undergone primary PCI with stent implantation within 12 h of symptom onset. Patients were treated with aspirin, heparin, and a P2Y12 inhibitor according to American College of Cardiology/American Heart Association practice guidelines.

Electrocardiography analysis. Standard 12-lead electrocardiogram was recorded before and 1 h after the procedure and at 1, 4, 12, and 24 months of follow-up at a paper speed of 25 mm/s and a sensitivity of 10 mm/mV. Pre- and post-procedural ECGs were obtained at the catheterization laboratory. At 4 and 24 months, an ECG was recorded on the same day as CMR.

The ECG measurements were performed in a blinded fashion without knowledge of the results of the CMR or earlier ECGs. In case of disagreement regarding the ECG interpretation, a consensus was reached by reading the tracing together. All measurements were digitally performed using ImageJ (Version 1.43u, National Institutes of Health 2010, Bethesda, Maryland), enabling up to 0.01 ms accuracy. Measurements included Q-wave depth and duration as well as R-wave amplitude. All 12 leads except for AvR were used in this analysis.

All ECGs were scored according to the 4 clinically available definitions of Q-wave MI (5,6,8–10) (see also Table 1). These data were used to determine which of the 4 definitions shows strongest correlation with infarct size.

Q-wave regression was defined as re-classification from initial Q-wave MI defined 1 h after PCI to permanent non–Q-wave MI at 24 months of follow-up. We excluded ECGs with QRS confounders, that is, left bundle branch block, left ventricular hypertrophy, Wolff-Parkinson-White syndrome. Patients with more than 2 missing ECGs or ECGs of poor quality (n = 9) were excluded from the analysis.

ABBREVIATIONS AND ACRONYMS

AUC = area under the curve

CI = confidence interval

CMR = cardiac magnetic resonance

ECG = electrocardiogram

LVEF = left ventricular ejection fraction

MVO = microvascular obstruction

PCI = percutaneous coronary intervention

ROC = receiver-operating characteristic

STEMI = ST-segment elevation myocardial infarction

Table 1. Multiple Definitions of Pathological Q Waves

"Classic" criteria	Q-wave with a duration ≥40 ms and/or a depth ≥25% of the R-wave in the same lead or the presence of a Q-wave equivalent
TIMI criteria	Q-wave ≥30 ms in 2 contiguous leads, any Q- or R-wave ≤10 ms and ≤0.1 mV in lead V_2, and R-wave ≥40 ms in V_1
Consensus 2000	Any Q-wave in leads V_1 through V_3, Q-wave ≥30 ms in leads I, II aVL, aVF, V_4, V_5, V_6
	The Q-wave changes must be present in any 2 contiguous leads, and be ≥1 mm in depth
Consensus 2007	Any Q-wave in leads V_2–V_3 ≥20 ms or QS complex in leads V_2 and V_3
	Q-wave ≥30 ms and ≥0.1 mV deep or QS complex in leads I, II, aVL, aVF or V_4–V_6 in any 2 leads of a contiguous lead grouping (I, aVL, V_6; V_4–V_6; II, III, and aVF)
	R-wave ≥40 ms in V_1–V_2 and R/S ≥1 with a concordant positive T-wave in the absence of a conduction defect

TIMI = Thrombolysis In Myocardial Infarction.

Cardiac magnetic resonance. CMR examination was performed at 4 ± 2 days after reperfusion and repeated after 4 and 24 months. Contrast-enhanced CMR was performed at baseline and 4 months. Patients were studied on a clinical 1.5-T scanner. Contiguous short-axis slices were acquired every 10 mm, covering the whole LV from base to apex using a segmented steady-state free precession pulse sequence. Late gadolinium enhancement images were obtained 10 to 15 min after administration of a gadolinium-based contrast agent (Dotarem, Guerbet, Villepinte, France) (0.2 mmol/kg) using a 2-dimensional segmented inversion recovery gradient echo pulse sequence, with slice position identical to the cine images, to examine infarct size and segmental transmural extent of infarction. Typical in-plane resolution was 1.4 × 1.7 mm², with slice thickness 5.0 to 6.0 mm (repetition time/echo time = 9.6/4.4 ms, flip angle 25°, triggering to every other heartbeat). The CMR data were analyzed using a dedicated software package (Mass 2008beta, Medis, Leiden, the Netherlands). CMR studies were analyzed blinded to ECG results and patient identity. On short-axis cine slices, the endocardial and epicardial borders were outlined manually on end-diastolic and end-systolic images. From these, LVEF was calculated. Infarct size was determined on the late gadolinium enhancement images as previously described using a standardized and pre-defined definition of hyperenhancement. Total infarct size was expressed as percentage of LV

mass (14). Transmural extent of infarction was calculated by dividing the hyperenhanced area by the total area of the pre-defined segment (%). Microvascular obstruction (MVO) was assessed using late gadolinium enhancement and defined as any region of hypoenhancement within the hyperenhanced infarcted area. This area was included in the calculation of total MI size. For analysis of regional myocardial function, each short-axis slice was divided into 12 equiangular segments to calculate wall thickening (in millimeters) of each segment by subtracting end-diastolic from end-systolic wall thickness. Myocardial segments were considered dysfunctional if segmental wall thickening was <3 mm (15).

Statistical analysis. Values are reported as mean ± SD or median (25th to 75th percentile) for continuous variables and as frequency with percentages for categorical variables. Unpaired Student *t* test and a Fisher exact test were used to compare differences between groups of continuous and categorical variables, respectively. Receiver-operating characteristic (ROC) curves were constructed to assess association of Q-wave MI distinction based on the 4 different criteria with infarct size. The comparison of areas under the ROC curves (AUC) was performed as described by DeLong et al. (16). Also, the differences of infarct size between Q-wave and non–Q-wave MI patients were assessed with the unpaired Student *t* test. Association of LVEF, LV dimensions, and infarct size in patients with Q-wave regression was compared with that of patients without pathological Q waves and patients with persistent Q waves. For this analysis, Q waves were classified according to the criteria that had the best association with infarct size. No adjustments for multiple comparisons were made. All statistical tests were 2-tailed, and a value of p < 0.05 was considered statistically significant. Calculations were generated by SPSS software (version 18.0 for Windows, SPSS, Chicago, Illinois).

RESULTS

During follow-up, 2 patients died due to noncardiac causes, and 5 patients presented with myocardial re-infarction. These patients were excluded from the final study population. Table 2 shows the clinical characteristics of the final study population. **Predictive value of different ECG criteria.** In Figure 1, ROC curves for the 4 different Q-wave criteria are shown for 1-h ECGs and the relation with infarct size. Association with infarct size was significantly better for Q-wave distinction based on the classic

Table 2. Clinical Characteristics of the Study Population (N = 184)

Demographics	
Age, yrs	56 ± 9
Male	156 (85)
Risk factors	
Diabetes mellitus	12 (7)
Known hypertension	55 (30)
Family history of coronary heart disease	90 (49)
Hypercholesterolemia	35 (19)
Current cigarette smoking	93 (51)
Angiography and infarct treatment	
Time from symptom onset to PCI, h	3.3 (2.2–4.6)
Infarct-related artery	
Left anterior descending artery	118 (64)
Right coronary artery	43 (23)
Left circumflex artery	23 (13)
Multivessel disease	46 (25)

Values are mean ± SD, n (%), or median (25th–75th percentile).
PCI = percutaneous coronary intervention.

Figure 1. ROC Curves Describing the Association With Infarct Size for the 4 Different Criteria for Q-Wave/Non–Q-Wave MI Based on ECG 1 h After PCI

Receiver-operating characteristic (ROC) curves for the 4 different Q-wave criteria are shown for 1-h ECGs and the relation with infarct size. Association with infarct size was significantly better for Q-wave distinction based on the classic criteria. AUC = area under the curve; CI = confidence interval; MI = myocardial infarction; PCI = percutaneous coronary intervention; TIMI = Thrombolysis In Myocardial Infarction.

	AUC	95% CI
Classic Criteria	0.71	0.60–0.82
2000 Criteria	0.57	0.47–0.68
TIMI Criteria	0.57	0.47–0.68
2007 Criteria	0.57	0.47–0.67

criteria (23% classified as Q-wave MI; AUC: 0.71, 95% confidence interval [CI]: 0.60 to 0.82) compared with the 2007 criteria (58% classified as Q-wave MI; AUC: 0.57, 95% CI: 0.47 to 0.67, p < 0.01) and 2000 and TIMI criteria (respectively, 55% classified as Q-wave MI; AUC: 0.57, 95% CI: 0.47 to 0.68, p < 0.01, and 53% classified as Q-wave MI; AUC: 0.57, 95% CI: 0.47 to 0.68, p < 0.01). Similar results were shown for infarct size at 4 months; AUC of the classic criteria was 0.69 (95% CI: 0.58 to 0.79) compared with the 2007 criteria (0.58, 95% CI: 0.48 to 0.68, p = 0.13) and 2000 and TIMI criteria (respectively, AUC: 0.59, 95% CI: 0.49 to 0.69, p = 0.14, and AUC: 0.60, 95% CI: 0.50 to 0.60, p = 0.14).

Difference in infarct size (% of LV mass) between patients with Q-wave and non–Q-wave MI according to the 4 different criteria are described in Table 3. We observed similar patterns for all other ECG time points as well as for infarct size at 4 months, also after correcting for infarct locations (data not shown).

Infarct size and LVEF in patients with Q-wave versus non–Q-wave MI. Patients with Q-wave MI according to the classic criteria had larger infarct size and lower LVEF on baseline CMR (respectively, 24 ± 10% LV mass and 37 ± 8%) compared with patients with non–Q-wave MI (17 ± 9% LV mass, p < 0.01, and 45 ± 8%, p < 0.001). Also at 4 months, this difference between patients with Q-wave and non–Q-wave MIs was observed, and for LVEF also at 24 months (Table 4).
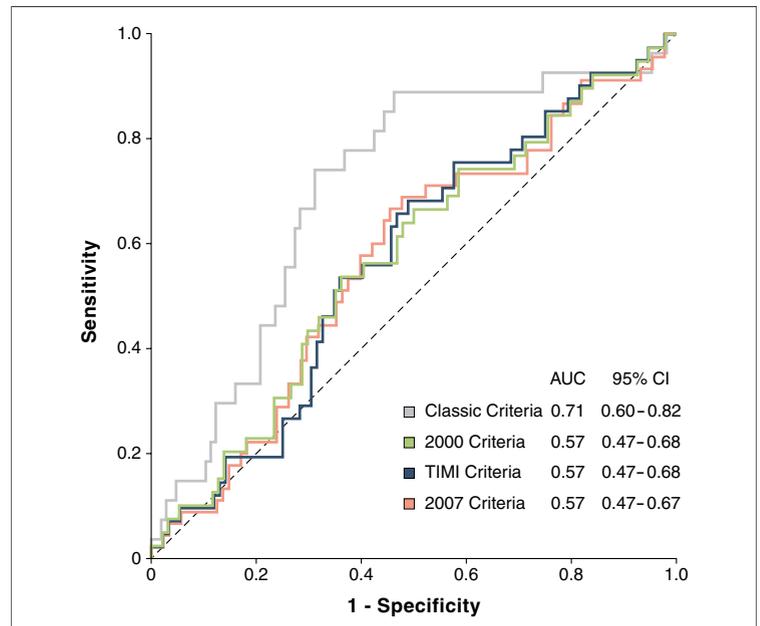
Largest improvement of LVEF in patients with Q-wave regression at 24 months. In patients with initial Q-wave MI, Q-wave regression (both assessed according to classic criteria) within 24 months of follow-up was present in 40% of the patients (n = 17). Ten patients had Q-wave regression between 1 h and 1 month post-PCI. Seven patients had Q-wave regression between 1 month and 4 months. At baseline, patients with Q-wave regression and patients with persistent Q-wave MI had comparable LVEF and infarct size. However, at 4 months, the Q-wave regression was accompanied by a decrease in infarct size and, at 24 months, with the greatest improvement of LVEF over time, com-

Table 3. Infarct Size (% of LV Mass) for Q-Wave MI and Non–Q-Wave MI as Assessed 1 h After PCI Comparing the 4 Widely Available Definitions

	Non–Q-Wave MI	Q-Wave MI	p Value
"Classic" criteria	17 ± 9	24 ± 10	<0.001
2000 criteria	18 ± 10	20 ± 10	0.28
TIMI criteria	18 ± 10	21 ± 10	0.16
2007 criteria	18 ± 10	20 ± 10	0.40

LV = left ventricular; MI = myocardial infarction; other abbreviations as in Tables 1 and 2.

Table 4. CMR Characteristics of the Study Population Split for Non–Q-Wave, Q-Wave MI, and Q-Wave Regression Groups According to the Classic Criteria

CMR Total (N = 184)	Non–Q-Wave MI (n = 141)	Persistent Q-Wave MI (n = 26)	Q-Wave Regression (n = 17)	Non–Q-Wave MI vs. Q-Wave MI p Value	Non–Q-Wave MI vs. Q-Wave Regression p Value	Q-Wave MI vs. Q-Wave Regression p Value
Infarct size, % of LV mass						
Baseline	17 ± 9	25 ± 5	24 ± 14	0.02	0.36	0.58
4 months	11 ± 7	17 ± 5	15 ± 7	0.02	0.03	0.86
LV ejection fraction, %						
Baseline	45 ± 8	37 ± 8	38 ± 7	<0.001	0.02	0.72
4 months	49 ± 9	40 ± 7	43 ± 10	<0.001	0.002	0.23
24 months	49 ± 8	40 ± 8	48 ± 11	<0.001	0.55	0.02
LV end-diastolic volume, ml/m²						
Baseline	96 ± 16	103 ± 15	100 ± 12	0.045	0.44	0.39
4 months	101 ± 17	115 ± 25	109 ± 30	0.01	0.20	0.56
24 months	102 ± 20	127 ± 32	106 ± 42	<0.001	0.50	0.10
LV end-systolic volume, ml/m²						
Baseline	53 ± 15	66 ± 14	60 ± 13	<0.001	0.28	0.46
4 months	52 ± 16	70 ± 21	65 ± 29	<0.001	0.03	0.46
24 months†	54 ± 19	78 ± 27	59 ± 38	<0.001	0.29	0.12
Presence of microvascular obstruction	71 (53)	24 (92)	9 (53)	<0.001	1.00	0.002
Segmental extent of transmurality, %	11 ± 12	22 ± 13	19 ± 15	<0.001	0.03	0.47

Values are mean ± SD or n (%).
  CMR = cardiac magnetic resonance; other abbreviations as in Table 3.

pared with both persistent Q-wave MI as well as non–Q-wave MI (Table 4 and Fig. 2). Infarcts in the Q-wave regression group were not only larger than in the non–Q-wave MI patients but also more transmural. Size and extent of the infarcts at baseline of Q-wave regression patients was comparable to persistent Q-wave MI. However, MVO as assessed on baseline CMR was more frequently present in persistent Q-wave MI patients than in patients displaying Q-wave regression (92% vs. 53%, p = 0.002). At 4 months, there were no patients with MVO.
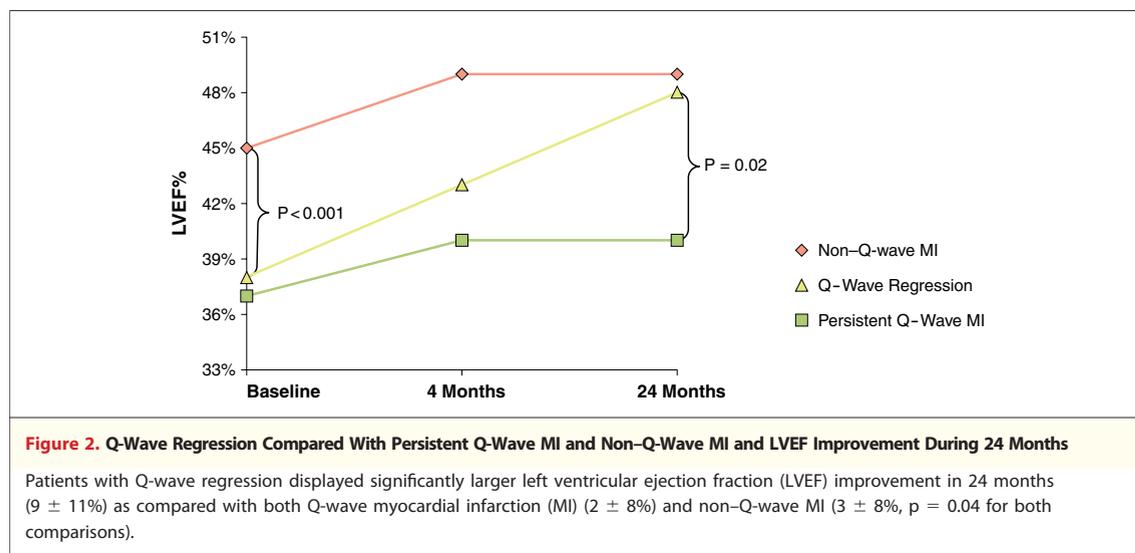
Regional function and recovery. In Figure 3, the regional wall thickness and function in dysfunctional segments are shown. Although wall thickening of the dysfunctional segments at baseline was similar between persistent Q-wave MI and Q-wave regression patients (respectively, 1.0 mm and 0.9 mm, p = 0.64), the improvement in wall thickening was larger in patients with Q-wave regression (2.8 mm compared with 2.0 mm for persistent Q-wave, p = 0.03, see Fig. 3A). This is in line with the differences in recovery of global LVEF between the groups. There was a decrease in end-diastolic wall thickness from baseline to 24 months follow-up in all 3 groups. However, this decrease was larger in the persistent Q-wave MI patients, (−2.7 mm compared with −1.9 mm for Q-wave regression and −1.6 mm for non–Q-wave MI, p = 0.002 and

p = 0.08, respectively (Fig. 3B). With regard to regional systolic function, no change from 24 months follow-up to baseline in end-systolic wall thickness in the Q-wave regression and non–Q-wave MI group was observed (−0.1 mm compared with 0.2 mm, p = 0.73). This was in contrast to a significant decrease in end-systolic wall thickness in the persistent Q-wave MI group (Fig. 3C) (−1.7 mm, p = 0.001 and p = 0.008, respectively). This resulted in the described changes in wall thickening during follow-up (Fig. 3D).

DISCUSSION

Association of infarct size on CMR was best when making Q-wave/non–Q-wave distinction based on the classic Q-wave criteria The Q-wave/non–Q-wave MI distinction is of clinical relevance, because Q waves are associated with a lower ejection fraction and a larger MI. Q-wave regression at 24 months was present in 40% of the patients with initial Q-wave MI and was associated with the largest reduction of infarct size and improvement of LVEF as assessed on CMR. Moreover, patients with persistent Q waves had more often (92%) MVO on baseline CMR.

Q-wave and incidence. We assessed Q-wave incidence in a STEMI population treated with primary PCI without a history of prior MI and subsequent

Figure 2. Q-Wave Regression Compared With Persistent Q-Wave MI and Non–Q-Wave MI and LVEF Improvement During 24 Months

Patients with Q-wave regression displayed significantly larger left ventricular ejection fraction (LVEF) improvement in 24 months (9 ± 11%) as compared with both Q-wave myocardial infarction (MI) (2 ± 8%) and non–Q-wave MI (3 ± 8%, p = 0.04 for both comparisons).

Q waves. In our study, the incidence of pathological Q waves was 58% 1 h after PCI, based on the 2007 criteria. The classic criteria were stricter, labeling 23% of the patients as having pathological Q waves. Other studies have provided wide ranges of Q-wave MI incidence, partly depending on which criteria were used. A recent study found an incidence of 57% using 2007 criteria, which is comparable to this study (4).

Q-wave and different criteria. The criteria for pathological Q waves have changed over the years. Jensen et al. (17) already addressed the redefinition of the Q-wave when comparing the classic criteria with the consensus document in 2000 to assess prior myocardial infarction. They found in 79 patients with and 77 patients without previous MI that the 2000 criteria were nonspecific, resulting in a high number of false-positive results. However, they only compared 2 definitions and used misquoted criteria from the 2000 consensus document. Nowadays, 4 different sets of criteria are used in daily clinical practice. To our knowledge, our study is the first to compare these different Q-wave criteria. We found that of these 4 different pathological Q-wave criteria, the distinction based on the classic criteria was the best predictor of infarct size on CMR. This is probably due to more strict criteria compared with the new criteria, thus resulting in a lower number of false positives.

Q-wave and prediction of LVEF and infarct size. In the present study, Q-wave MI was associated with larger infarct size and lower LVEF, both at baseline as well as at follow-up. This confirms earlier studies showing larger MIs as assessed with CMR in Q-wave MI patients (2–4). However, to our knowledge, we are the first to assess the Q-wave/non–Q-wave MI dis-

tinction in a large population using CMR up to 24 months of follow-up, showing that the Q-wave distinction remains clinically relevant.

Q-wave and regression. Though Q waves are considered to be permanent stigmata of infarction, several large studies in the thrombolytic era have shown they disappear in as much as 15% of all cases (11–13). The disappearance of Q waves has even been documented after successful myocardial revascularization by coronary artery bypass grafting (18). In the era of primary PCI, there has been only 1 report to date where Q-wave regression was assessed (19). Q-wave regression in ≥2 leads occurred in approximately 10% to 20% of the study population. In that study, conducted by Iwasaki et al. (19), the researchers did not find a correlation between the incidence of Q-wave regression and LVEF, LV volumes, or regional wall motion. This was, however, investigated in a small population of 94 patients, of which only 47 patients were treated with primary PCI.

CONCLUSIONS

In the present study, we show that Q-wave regression is associated with LVEF improvement. Thus, for the first time, we now show that there is indeed a direct relationship between functional LV recovery after MI and the disappearance of initial Q waves.

A new ventricular conduction disturbance may alter activation pathways and mask prior Q waves. Similarly, a second MI involving the contralateral ventricular wall may cause an apparent regrowth of R waves over the area of previous infarct. However, we excluded patients with a clinical evidence of re-
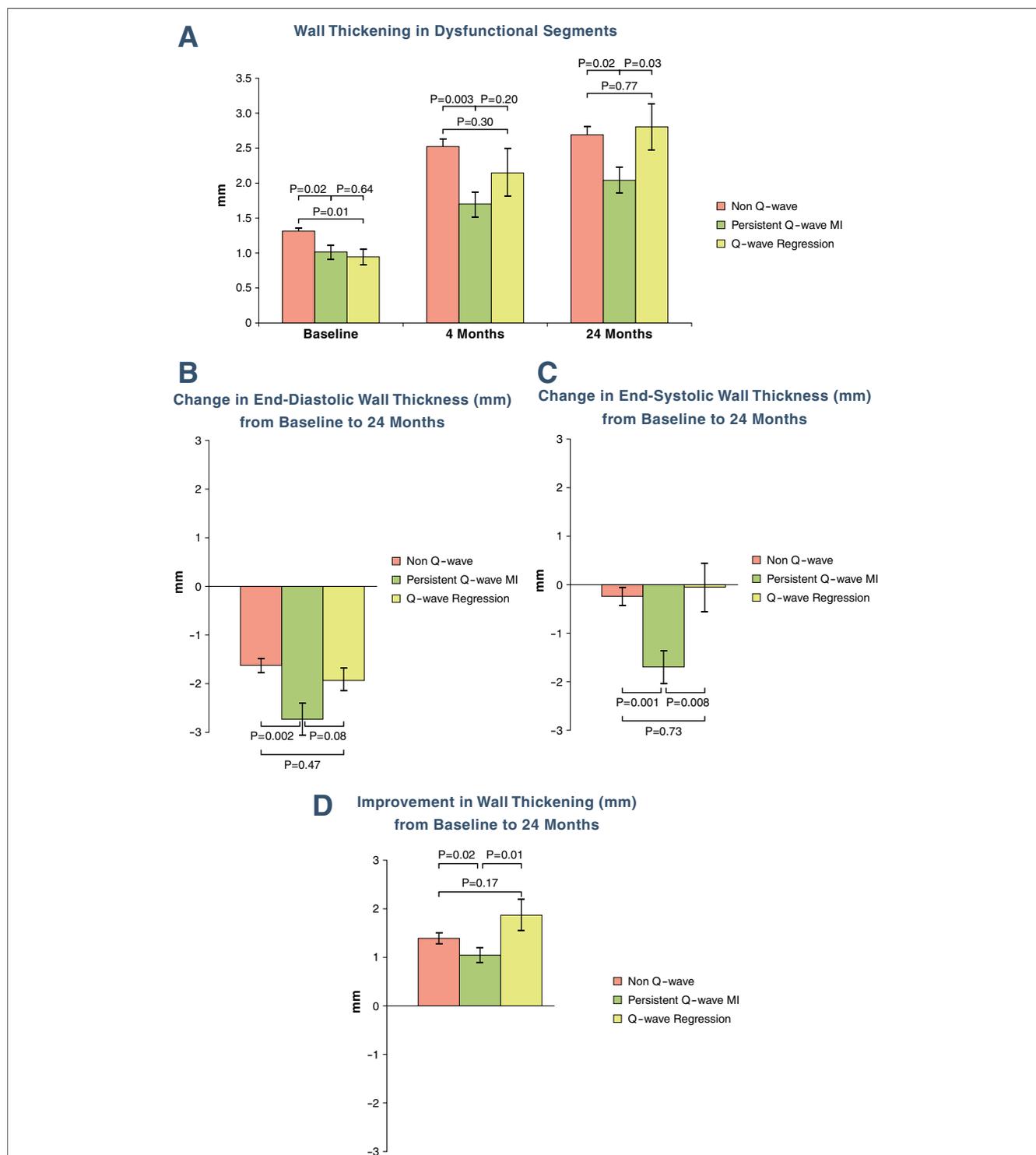
Figure 3. Regional Wall Thickness and Function in Dysfunctional Segments

(A) Wall thickening in dysfunctional segments at baseline, 4 months, and 24 months stratified to Q-wave regression, persistent Q-wave myocardial infarction (MI), and non–Q-wave MI. **(B)** Change in end-diastolic wall thickness (mm) stratified to Q-wave regression, persistent Q-wave MI, and non–Q-wave MI. **(C)** Change in end-systolic wall thickness (mm) stratified to Q-wave regression, persistent Q-wave MI, and non–Q-wave MI. **(D)** Improvement in wall thickening (mm) stratified to Q-wave regression, persistent Q-wave MI, and non–Q-wave MI.

infarction, and also the marked improvement of LV function in this specific group of patients suggests differently. We observed that the presence of MVO was strongly related to persistence of Q waves. We and others have previously shown the correlation between MVO and LV functional recovery (20,21). Apparently, MVO reflects, not only MVO, but also severity of myocardial damage and a lower capacity to regain function. True myocardial healing also cannot be excluded, although the regenerative capacity of the myocardium is known to be very limited. Nevertheless, Q-wave regression points out a separate group of patients with a larger than average capability to recover LV function after PCI treated acute MI.

Study limitations. The Minnesota criteria have also been developed for the presence of Q waves. These criteria are based on computer algorithms and developed for epidemiological studies and clinical trials. In our study, we did not use these criteria because we focused on readily available definitions that can be used in daily clinical practice. Also, this study is limited by a small sample size, especially in the group with Q-wave regression. However, despite the limited sample size, we have observed statistically significant and clinically relevant differences between patients without Q waves, persistent Q waves, and Q-wave regression.

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