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The prognostic role of electrocardiographic left ventricular mass assessment for identifying PCI-treated patients with acute ST-elevation myocardial infarction at high risk of unfavourable outcome

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Abstract

Background: *In prognostic terms, evaluation of an ECG recording in acute myocardial infarction (AMI) appears to be inferior to echocardiographic (ECHO) assessment of left ventricular remodelling and the activities of cardiac enzymes and certain hormones. It was our hypothesis that, in the era of interventional treatment of AMI, some ECG parameters are still valid for the purpose of risk stratification.*

Methods: *A total of 66 consecutive patients with AMI (43 male and 23 female, with a mean age of 61 ± 11 years) were treated with primary percutaneous coronary intervention (PCI). In each patient ECG and ECHO examinations were performed within 5–7 days of admission for the detection of left ventricular hypertrophy (LVH). In further analysis the following ECG-based LVH parameters were taken into consideration: Sokolov-Lyon voltage duration (SLVd), Cornell voltage duration CVd), 12-lead QRS voltage duration (12QRSVd), their product with QRS duration and an ECG index of left ventricular mass ($LVMI_{ECG}$). Patients were followed for 6 months. The combined end-point included death, infarction, a need for prompt coronary intervention and hospitalization for heart failure.*

Results: *The combined end-point was observed in 16 patients (24.2%). Survival analysis revealed that the most important prognostic factors were associated with a prolongation of the QRS duration. Increased SLVd was found in 43% of the patients with events compared to 14% in those without them ($p < 0.01$), CVd in 43%* vs. *12% ($p < 0.05$), 12QRSVd in 81%* vs. *44% ($p < 0.05$) and $LVMI_{ECG}$ in 75%* vs. *26%, $p < 0.001$). There was no evidence for a difference in Cornell voltage. Univariate logistic regression indicated a 4-fold to 8-fold increase in the risk of events associated with abnormal SLV, SLVd or $LVMI_{ECG}$. Multivariate Cox analysis showed that the LVH presence in the ECG, defined as an increased SLVd product or increased $LVMI_{ECG}$, was an independent predictor of cardiovascular events after AMI.*

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Conclusions: *In the era of interventional treatment of AMI, the ECG features of left ventricular hypertrophy carry independent significant prognostic information.* (Cardiol J 2007; 14: 347–354)

Key words: left ventricular mass, left ventricular hypertrophy, electrocardiography, QRS complex, echocardiography, coronary artery disease, acute ST-elevation myocardial infarction, prognosis

Introduction

The prognostic use of routine electrocardiogram (ECG) in patients with an acute myocardial infarction (AMI) currently appears to have been superseded by biochemical markers and echocardiographic data [1], despite the large body of evidence accumulated over decades indicating that many ECG variables carry information about in-hospital and long-term outcome [2–6]. Early studies indicated a greater threat to patients who presented with Q-wave infarction [2], a greater QRS score [3, 4], a slow ST-segment resolution rate [5, 6], prolonged QT and increased QT dispersion [7, 8] or sustained T-wave abnormalities [9].

In many of these studies, however, the presence of left ventricular hypertrophy (LVH) was deemed to be an exclusion criterion, despite the fact that LVH was found to be a factor multiplying the risk for subsequent events in the AMI-patient population [10–12]. In fact, these studies were conducted before the advance of myocardial reperfusion techniques. Thus the significance of the presence of LVH requires verification in AMI-patients treated with the percutaneous coronary interventions (PCI) currently commonly applied. Accordingly, we undertook this study to solve the question of whether ECG-based LVH detection might be helpful in distinguishing AMI-patients at high risk of an unfavourable outcome.

Method

Study population

A total of 66 consecutive patients with ST-elevation acute myocardial infarction (STEMI) treated by means of direct coronary stenting were included in the study. There were 43 males and 23 females with a mean age of 61 ± 11 years. They entered the study if the following inclusion criteria were met: first STEMI, successful coronary stenting (TIMI coronary flow grade 3 after intervention), uncomplicated in-hospital stay, stable sinus rhythm and the granting of informed consent. Patients who needed circulatory support, with conditions that limited LVH detection (intraventricular conduction disturbances, implanted cardiac stimulator or an inadequate echocardiographic window) or who suffered from an illness which might limit short-term (6-month) survival were excluded. The clinical characteristics of the patients are given in Table 1.

Left ventricular hypertrophy detection on ECG

Routine 12-lead surface ECGs (ECG, GE Medical Systems, CASE v.4.1) were recorded before hospital discharge (usually 5–7 days after the coronary intervention) for all the patients in order to determine left ventricular mass (LVM_{ECG}) and the presence of LVH_{ECG} features. The median cardiac cycle complexes were collected automatically and displayed on a screen using the magnifying function with a gain of up to 8 cm/1 mV and a paper speed of 200 mm/s. The QRS complex parameters were measured manually with a calliper to the nearest 1 ms and voltage was measured to the nearest 4 μV. All measurements were performed in accordance with the Common Standards in Electrocardiography Working Party recommendations [13] by an experienced investigator (MS) who had no knowledge of the patients' clinical data and echocardiographic findings. The following ECG variables and indices were obtained from 12 simultaneously recorded leads: QRS complex duration (QRSd, ms), average 12-lead time to maximal deflection (TMD, ms), average 12-lead QRS complex voltage (12QRSV, mV), calculated as a sum of absolute voltages of all deflections within 12 QRS complexes divided by the number of leads, and the average product of 12-lead QRS voltage and duration (12QRSVd, mV/ms). Commonly recommended ECG criteria were also calculated: Sokolov-Lyon voltage (SLV, mV) as a sum of the S-wave voltage in the V_1 or V_2 leads and the R-wave voltage in V_5 or V_6 (whichever higher), Cornell voltage (CV, mV) as a sum of the R-wave voltage in the aVL lead and the S-wave voltage in the V_3 lead. A correction for female gender was made by adding 0.6 mV to the calculated CV. Their voltage-duration products (SLVd and CVd) were also calculated [14–16].

The LVM_{ECG} was calculated by analogy to echocardiographically-determined LVM, according

Table 1. Clinical characteristics of the study group.

Parameter	All patients (n = 66)	With event (n = 16)	Event-free (n = 50)
Age (years)[#]	61 ± 10	68 ± 9	59 ± 10
Sex (male)	43 (65)	8 (50)	35 (70)
Body mass index [kg/m^2]	27.1 ± 4.0	27.1 ± 3.3	27.1 ± 4.2
Obesity (BMI > 30 kg/m^2)	13 (20)	4 (25)	8 (16)
Risk factors			
Smoking (current or in the past)	47 (71)	11 (69)	36 (72)
Hyperlipidemia	49 (74)	12 (75)	37 (74)
Arterial hypertension	39 (59)	12 (75)	27 (54)
Diabetes mellitus or OGI	16 (24)	6 (38)	10 (20)
Family history	16 (24)	4 (25)	12 (24)
STEMI characteristics			
Location:			
anterior	21 (31)	7 (44)	14 (28)
inferior	38 (58)	8 (50)	30 (60)
other	7 (11)	1 (6)	6 (12)
Infarct-related artery:			
LAD/Diag1	18 (27)	7 (44)	11 (22)
RCA	31 (47)	7 (44)	24 (48)
LCx/OM	17 (26)	2 (8)	15 (30)
Killip class > II*	6 (9)	4 (9)	2 (4)
Systolic blood pressure [mm Hg]	119 ± 17	112 ± 24	122 ± 14
Heart rate [bpm]	72 ± 11	76 ± 13	71 ± 10
Echocardiographic data (at discharge)			
LVEF [%]	47 ± 9	48 ± 9	47 ± 9
LVEF < 40%	9 (13)	2 (13)	7 (14)
LVEDD [cm]	5.1 ± 0.6	5.3 ± 0.7	5.0 ± 0.5
LVEDD > 5.5 cm	13 (20)	5 (31)	8 (16)
LVMI [g/m^2][§]	105.1 ± 24.1	122.7 ± 30.2	99.4 ± 18.8
LVMI > UNL	16 (24)	6 (38)	10 (20)
Medications (at discharge)**			
Beta-blocker	61 (92)	14 (88)	47 (96)
ACEI and/or ARB	64 (96)	15 (94)	49 (98)
Antiarrhythmics	5 (8)	3 (19)	2 (4)

SD — standard deviation, OGI — oral glucose intolerance, STEMI — ST-segment elevation acute myocardial infarction, LAD — left anterior descendent branch of the left coronary artery, Diag — diagonal branch of the LAD, RCA — right coronary artery, LCx — left circumflex branch of the left coronary artery, OM — obtuse marginal branch of the LCx, LVEF — left ventricular ejection fraction, LVEDD – left ventricular end-diastolic diameter, LVMI — index of the left ventricular mass, UNL — upper normal limit, ACEI — angiotensin converting-enzyme inhibitor, ARB — angiotensin AT1-receptor inhibitor; **other medications (aspirin, statins) were administered to all patients. Data presented as mean ± 1 SD or number (%) as appropriate. Statistics: discrete data were compared using Student's t test for unpaired comparisons, numerical data were compared using the two-sided Fisher exact test. *$p < 0.05$, [#]$p < 0.01$, [§]$p < 0.001$ — significance of the difference between patients with and without an event

to the following formula: $LVM_{ECG} = \{[(2TMD) + +(QRSD/\pi)^3 - (QRSD/\pi)^3]\} \times 0.0001$ [ms^3], and indexed to body surface area (BSA), providing the $LVMI_{ECG}$ as [LVM_{ECG}/BSA, ms^3/m^2], introduced by the authors and described in detail elsewhere [17].

Left ventricular hypertrophy detection on ECHO

Transthoracic echocardiographic examinations [Sonos 2500, Hewlett-Packard] were performed on all the patients on the same day by an experienced echocardiographer (BK) blind to other data. LV anatomy and function were assessed in accordance with current American Society of Echocardiography recommendations [18] to obtain the following parameters:

— LV diastolic and systolic diameter, and inferior LV wall and interventricular septum thickness from the left parasternal long-axis view (all in centimetres);
— LV ejection fraction, calculated according to Simpson's rule obtained from an apical four-chamber view (%),

Table 2. ECG left ventricular mass determinants depending on outcome.

Parameter	With event (n = 16)	Without event (n = 50)	Significance*
Heart rate [bpm]	71 ± 9	72 ± 12	NS
QRS duration [ms]	109 ± 10	102 ± 6	< 0.001
QTc max [ms]	463 ± 28	446 ± 32	NS
QT dispersion [ms]	32 ± 10	29 ± 11	NS
SL voltage [mV]	3.30 ± 1.22	2.51 ± 0.83	< 0.05
SL v/d [mV/ms]	364 ± 148	258 ± 90	< 0.05
Cornell voltage [mV]	2.12 ± 0.83	1.94 ± 0.59	NS
Cornell v/d [mV/ms]	233 ± 99	199 ± 61	NS
12QRS voltage [iV]	1.46 ± 0.36	1.21 ± 0.30	NS
12QRS v/d [mV/ms]	160 ± 45	123 ± 34	< 0.05
LVM_{ECG} [ms^3]	252 ± 81	194 ± 49	< 0.01
$LVMI_{ECG}$ [ms^3/m^2]	134 ± 41	105 ± 26	< 0.005

SL — Sokolov-Lyon index, 12QRS — the average of 12-lead QRS, LVM — left ventricular mass, v/d — voltage-duration product; *Kolmogorov--Smirnov test

— LVM_{ECHO}, calculated according to the corrected ASE-cube formula [18]: LVM [g] = 0.8 [1.04 {(LVEDD + IVSThd + LVPWThd)^3 – –LVEDD^3}] + 0.6, and indexed for BSA ($LVMI_{ECHO}$). LVH was diagnosed if $LVMI_{ECHO}$ exceeded 117 g/m^2 in men and 104 g/m^2 in women [19].

Follow-up

Patients were observed for a period of 6 months. The following events were noticed: death (for any reason), myocardial infarction, a need for urgent coronary intervention, hospitalization for heart failure or a cerebrovascular event. The above-listed criteria constituted a combined end-point of the study.

Statistics

Numerical data are presented as means + 1 SD, while categorical data are given as number and percentage, as appropriate. Between-group comparisons were made using the Kolmogorov-Smirnov test. Proportions were tested by means of the Fisher exact test. Between-variable correlations were tested by means of the Pearson-moment correlation analysis. Test accuracy was compared using ROC curve analysis. Logistic regression analysis was used to identify a significant association between the variables analysed and event occurrence. Cox regression hazard analysis was used to identify independent predictors. For all tests a p-value < 0.05 was considered significant.

Results

The combined end-point was noticed in 16 patients (24.2%). There was no death during the 6-month follow-up. Other events observed were as follows: recurrent myocardial infarction in one patient, unstable angina with a need for coronary intervention (either percutaneous or surgical) in 8 patients, neurological complications in 4 patients and heart failure development in 3 patients. ECG features depending on outcome are presented in Table 2, in general ECG variables that are considered to reflect LVM and form the basis for LVH in clinical settings. Among these, the most significant differences between patients with and without an unfavourable outcome in this respect were found for the time-dependent variables.

On the basis of the receiver-operator curve function the following cut-off values of the ECG LVH descriptors were further used: SLV > 3.4 mV, SLVd > 343.7 mV/ms, CV > 2.2 mV, CVd > 260 mV/ms, 12QRS voltage > 1.23 mV, 12QRS voltage/duration product > 123 mV/ms and $LVMI_{ECG}$ > 115 ms^3/m^2. The proportion of events associated with different ECG criteria is given in Table 3, while their statistical performance in presented in Table 4. For a given cut-off value a different event rate was observed. The concordance of the applied ECG criteria with echocardiographically-determined LVH is presented in Figure 1. Correlations between certain ECG variables and LVMI determined echocardiographically are presented in Figure 2.

The results of univariate logistic regression analysis indicated that the LVH ECG features most predictive for an unfavourable outcome were SLVd product and $LVMI_{ECG}$ (Table 5). This model allowed 90% of patients to be classified as without events and 50% as patients with events. Both SLVd product and $LVMI_{ECG}$ appeared as predictors of cardiovascular events independently of age and LV function

Table 3. Proportion of events depending on ECG variables.

Variable	Events (n = 16)	No events (n = 50)	p*
SL voltage > 3.4	8 (50)	8 (16)	0.0014
SL v/d > 343.7	9 (43)	7 (14)	0.0015
Cornell voltage > 2.2	9 (56)	14 (28)	0.0682
Cornell v/d > 260	7 (43)	6 (12)	0.0104
12QRS voltage > 1.23	12 (75)	21 (42)	0.0424
12QRS v/d > 123	13 (81)	22 (44)	0.0109
$LVMI_{ECG}$ > 115	12 (75)	13 (26)	0.0008

*Fisher's exact test (2-sided). Cut-off values of ECG variables as determined on the basis of ROC curve analysis. Abbreviations: see Table 2.

Table 4. Statistical performance of ECG variables in the prediction of events.

Variable	Sensitivity	Specificity	PPV	NPV	RR
S-L voltage > 3.4	61.5	84.9	50.0	90.0	3.1
S-L v/d > 343.7	56.2	86.0	56.2	86.0	3.1
Cornell voltage > 2.2	39.3	83.7	56.2	72.0	2.0
Cornell v/d > 260	53.8	83.0	43.8	88.0	3.6
12QRS voltage > 1.23	36.4	87.9	75.0	58.0	1.8
12QRS v/d > 123	37.1	90.3	81.3	56.0	1.8
$LVMI_{ECG}$ > 115	48.0	90.2	75.0	74.0	2.9

PPV — positive predictive value, NPV — negative predictive value, RR — risk ratio. Other abbreviations: see Table 2.

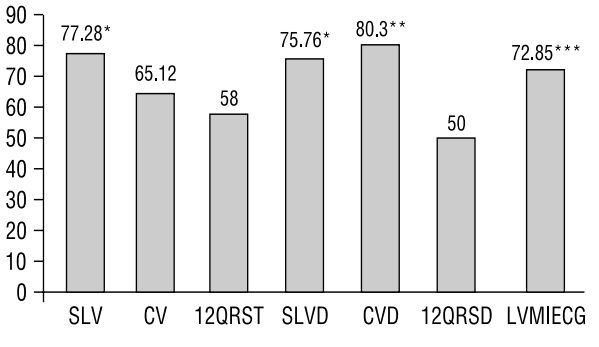

Figure 1. Concordance between ECG variables and echocardiographically-determined left ventricular hypertrophy (LVH). Bars represent the proportion of positive and negative LVH on ECG and echocardiography. Abbreviations: see "Method" section; *p < 0.05, **p < 0.01, ***p < 0.001, two-sided Fisher exact test.

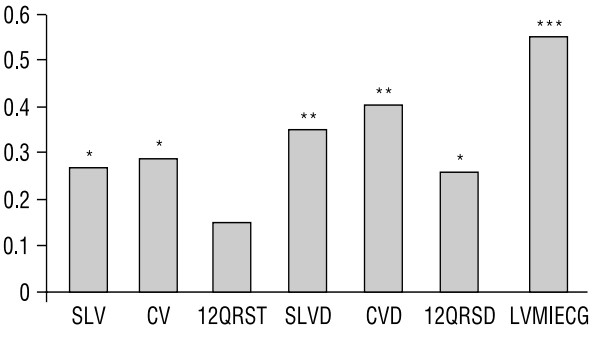

Figure 2. Coefficients of correlation between ECG variables and echocardiographically-determined LV mass index. Bars represent Pearson moment coefficients of correlation between a given ECG parameter and left ventricular mass index value. Abbreviations: see "Method" section; *p < 0.05, **p < 0.01, ***p < 0.001.

Table 5. Univariate logistic stepwise regression for the ECG variables examined and cardiovascular events during follow-up.

Variable	Odds ratio for event (95% CI)	p
S-L voltage-duration product	5.70 (1.43–22.68)	0.0134
Left ventricular mass index (ECG)	1.02 (1.00–1.04)	0.0413

Other ECG parameters did not enter the model.

Table 6. Predictors at 6 months of cardiovascular events (Cox hazard regression)

Parameter	Univariate		p	Multivariate*		p
	HR	95% CI		HR	95% CI	
SLV > 3.4	6.7	2.4–18.6	0.0003	–	–	–
SLVd > 343.7	7.8	2.8–22.3	0.0001	3.7	1.1–12.3	0.0335
CV > 2.2	2.5	0.95–9.8	NS	–	–	–
CVD > 260	4.1	1.5–11.1	0.0058	–	–	–
12QRSV >1.23	3.7	1.2–11.6	0.0224	–	–	–
12QRSVD >123	4.5	1.3–15.6	0.0199	–	–	–
$LVMI_{ECG}$ > 115	7.2	2.3–22.5	0.0008	3.9	1.0–15.1	0.0462

*Forward-stepwise regression, – not entered Cox regression model. Age, LVEF and LVEDD did not enter the model; HR — heart rate. Other abbreviations: see Table 2.

assessed echocardiographically. The respective hazard ratios are presented in Table 6.

Discussion

Main findings

In the present study we showed that ECG features of increased LVM, albeit only moderately sensitive, were independently associated with a clinical outcome over a 6-month period in PCI--treated patients with STEMI. We found the "classical" voltage LVH criteria to be of limited value in these patients. Taking QRSd into account meaningfully improved their statistical performance. Interestingly, a voltage-independent LVMI, proposed by the authors [17], which describes abstract time conditions [m^3] that are required for the entire cardiac muscle to be depolarized, was not found to be inferior to the SLVd product and appeared as a predictor of risk independently of age and LV ejection fraction. The authors previously found that $LVMI_{ECG}$ significantly correlated with LVM estimated echocardiographically [17].

Left ventricular hypertrophy and post-AMI prognosis

Almost 30 years ago Pohjola et al. [20] noticed that the presence of abnormal P terminal force was associated with a 4.7 times greater risk of mortality in a large cohort of patients with AMI over a 5-year follow-up. Although the authors considered the P-wave terminal force of –0.03 mm/ms or greater to be a feature of heart failure, one has to acknowledge that the same ECG pattern had been treated over decades as one of the early signs of LVH and was thus included in various LVH point-scores [21]. Several studies brought proof of a significant association between LVH on ECG and short-term and long-term post-myocardial infarction mortality in large cohorts of fibrinolyzed patients with either ST-elevation or non-ST-elevation AMI [11, 22–25], despite less sensitive ECG-voltage LVH criteria being used in these studies. More recent studies indicate the advantage of time-dependent ECG criteria, such as a prolongation of the QRSd [26]. Our finding of an independent association between $LVMI_{ECG}$, the index entirely based on time-related measurements, and short-term post-myocardial infarction outcome seems to support this observation.

QRSd and post-myocardial infarction prognosis

The association between QRSd prolongation and an unfavourable outcome in post-myocardial infarction patients has been the subject of recent studies [26–29]. Brilakis et al. [27] showed an independent association between a QRS prolongation ≥ 100 ms and in-hospital and overall mortality in patients with non-ST-elevation MI [26]. Other authors found a 4-fold greater risk of cardiac death in MI patients with QRS prolongation ≥ 120 ms. Importantly, in the Valsartan in Acute Myocardial Infarction (VALIANT) study, a prolongation of QRSd previously within normal limits was found to be a marker of increased risk of cardiovascular death, sudden death and the development of heart failure, although this did not possess independent prognostic value [28]. A recent analysis of the Danish Investigations and Arrhythmias and Mortality on Dofetilide (DIAMOND) study indicated that the prognostic value of QRSd is greater in post-myocardial infarction patients with LV dysfunction than in patients with heart failure. A 10 ms increase in QRSd was associated with a 6% increased risk of death over a 10-year period [29].

Accuracy of ECG measurements

Numerical ECG recording opened the opportunity for accurate ECG wave voltage and duration

measurements, and has recently been recommended [30]. The precision of the QRS determination used in the present study was far greater than in many previous studies. In our previous work we documented the fact that the measurement error for $LVMI_{ECG}$ attained approximately 1.5%, which corresponded to a value of ~1.8 ms^3/m^2 [17]. Such a low measurement error cannot be achieved with the use of a routine paper speed of 25–50 mm/s and gain of 1 cm/1 mV.

Other findings

Other interesting findings emerged from our study. Firstly, the authors found that an increased SLVd product and increased LVMI bore prognostic information for patients with a relatively preserved LV function and normal or only moderately prolonged QRS complex (< 120 ms). This finding should be considered significant, as the probability of influence of intraventricular conduction disturbances on the results of the study had been minimized by chosen range of the QRS duration. As early coronary interventions limit both myocardial injury and the development of significant intraventricular conduction delay, the proportion of patients with a QRSd within the 80–120 range would increase. Therefore distinguishing AMI-patients at higher risk for an unfavourable outcome among those with a relatively short QRS complex is a challenge, and our proposals help to meet this.

Secondly, in the group of patients with STEMI examined we were unable to confirm the advantage of the Cornell LVH criterion, currently commonly considered as more sensitive and specific for LVH diagnosis [14]. It has to be borne in mind that this criterion was developed in quite a different population of patients (those with hypertension) and for different purposes (mainly epidemiological) and, as far as the authors know [16], the Cornell University criterion has never been verified in patients with STEMI. Despite a somewhat greater concordance between an echocardiographically-determined increased LVM and the Cornell LVH criterion, the latter was not demonstrated be an independent risk predictor in our group of STEMI patients. One of the possible explanations is that the presence of myocardial scar reflected by the Q-wave appearance in the precordial V_1–V_3 leads might provide an erroneous calculation of SLV or the Cornell LVH index.

Interestingly, a discrepancy between LVH detection and the correlation with the LVMI was found for several commonly used voltage-dependent ECG LVH-criteria. As the determinants of the QRS voltage on the surface ECG are different and more important than upon echocardiogram, a voltage-independent ECG index, such as $LVMI_{ECG,}$ seems to be more promising, as indicated by the most significant, although still only moderate, correlation with $LVMI_{ECHO}$.

Limitations

The authors are aware that exclusion criteria can weaken the significance of findings and so the observations made and conclusion drawn cannot be extrapolated into the entire PCI-treated STEMI population. Furthermore, the cut-off values used should not be treated as limit values for LVH diagnosis, as they were chosen on the basis of ROC curve analysis with respect to the occurrence of an unfavourable outcome rather than LVH itself, despite these values lying within close proximity to the accepted ones [14]. We also kept in mind that echocardiography cannot be further considered as the "gold standard" for LVM determination in an era of ever more sophisticated and accurate methods such as nuclear magnetic resonance [31]. Nevertheless its use in this study was justified by the general application of echocardiography in clinical settings. Lastly, use of the QRSd-dependent index as a surrogate of LVM has its own limits related to the presence of ischemia and fibrosis, especially in the AMI setting. However, these two processes, both known to prolong and disturb the intraventricular conduction, are themselves important features of LVH and are initiated well before LVM increases enough to be diagnosed by means of echocardiography.

Conclusion

In an era of interventional treatment of acute myocardial infarction, the ECG features of left ventricular hypertrophy carry independent significant prognostic information.

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