

**MYOCARDIAL INFARCTION** 

# Utility of Contrast-Enhanced Cardiovascular Magnetic Resonance (CE-CMR) to Assess How Likely is an Infarct to Produce a Typical ECG Pattern

Juan Manuel Cino,<sup>1</sup> Sandra Pujadas,<sup>2</sup> Francesc Carreras,<sup>2</sup> Iwona Cygankiewicz,<sup>1</sup> Ruben Leta,<sup>2</sup> Mariana Noguero,<sup>2</sup> Xavier Garcia-Moll,<sup>2</sup> Tony Bayés Genís,<sup>2</sup> Guillem Pons-Lladó,<sup>2</sup> and Antoni Bayés de Luna<sup>1</sup>

Institut Catalá de Ciencies Cardiovasculars, Hospital Sant Pau, Sant Antoni M, Claret 167, CP 08025, Barcelona, Spain,<sup>1</sup> Servei de Cardilogia, Hospital de Sant Pau, Barcelona, Spain<sup>2</sup>

# ABSTRACT

Objectives: For over 50 years, Q-wave myocardial infarction (MI) location has been based on pathologic ECG studies. Although contrast-enhanced magnetic resonance (CE-CMR) is currently the "gold standard" technique for location and quantification of necrotic areas, we found no large study in the literature devoted to establish which ECG patterns corresponds to different MI location detected by CE-CMR. We hypothesized that CE-CMR would be very accurate for evaluating different ECG patterns and its sensitivity (SE) and specificity (SP) for locating MI in different LV areas. Methods and results: CE-CMR/ECG correlation was studied in 48 patients who presented a first MI due to acute coronary syndrome (ACS) with ST-segment elevation and in whom CE-CMR was performed in chronic phase. We evaluated the ECG patterns that best correlated with the 7 prespecified necrotic areas assessed by CE-CMR, 4 in anteroseptal zone (septal, apical/anteroseptal, extensive anterior, and limited anterolateral) and 3 in inferolateral zone (inferior, lateral and inferolateral). The global concordance between CE-CRM and ECG was of 75% and 7 ECG patterns were stablished. Conclusion: The capacity of CE-CMR to detect ECG patterns for necrotic area location presents highly acceptable concordance. Thanks to CE-CMR, we defined 7 ECG patterns for MI detection according to the 7 areas of the LV studied. The areas that present more cases with normal ECG are limited anterolateral and the areas of the inferolateral zone.

#### INTRODUCTION

Q-wave myocardial infarction (MI) location based on pathologic ECG studies conducted by Myers in the 1940s (1–2) became with minor changes, the most popular classification of Q wave MI (3–9). According to this classification, the presence of Q-waves in V1-V2 corresponds to septal MI, in V3-V4 to anterior MI, in V5-V6 and/or I-VL to low and high lateral MI, respectively, in II, III and VF to inferior or diaphragmatic MI, and R-(RS) in V1–V2 a mirror pattern of posterior infarction. However, clear evidence exists of the limitations of this strict classification owing to difficulties in correlating precordial leads with affected walls (precordial electrodes are often not well placed) and to changes in the precordial lead-heart wall relationship in subjects with different body builds (10–12). Furthermore, a large amount of information has been obtained from electrophysiological (13), hemodynamic, and angiographic studies (14–16) and, particularly, correlations with imaging techniques (17–19). Contrast-enhanced cardiovascular magnetic resonance (CE-CMR) has become the "gold standard" imaging technique for identifying the area of necrosis (20–24). Therefore, it is the ideal technique for assessing which ECG patterns better match the different infarcted areas detected by CE-CMR. The results of this study are presented herein.

## MATERIAL AND METHODS

CE-CMR and ECG findings were analyzed in 48 patients (40 men, 8 women, mean age: 62 yrs) in the chronic phase of

Keywords: Contrast-Enhanced Magnetic Resonance, Electrocardiography, Q Wave Infarction Correspondence to: A. Bayés de Luna Catedrático de Cardiología de la UAB Institut Catalá de Ciencies Cardiovasculars (ICCC) Sant Antoni M Claret 167, CP 08025, Barcelona, Spain email: abayesluna@santpau.es

MI. All had presented an ST elevation ACS. All patients were reperfused, with fibrinolytic or interventional therapy. Coronary angiography or multislice scanning was performed in 90% of cases.

## ECG recordings

Standard 12-lead ECGs were recorded at 25 mm/s speed and 10 mm/1 mV voltage in the chronic phase of MI ( $\geq$  3 weeks post MI). The ECG recordings were reviewed by 2 independent investigators blinded to clinical and CMR data. In cases of discrepancy, the final decision was made by a third investigator. MI was diagnosed according to the following ECG criteria (3, 4, 9, 25, 26) :1) any Q-wave  $\geq$ 30 ms in inferior/lateral leads; 2) Q wave  $\geq$ 40 ms in I, VL; 3) Q-wave in  $\geq$ 2 contiguous precordial leads; or 4 any Q-wave in V1-V2 or R wave  $\leq$ 0.1 mV in V2. The presence of RS in V1, R in V1  $\geq$ 40 ms, and "qr" or low voltage "r" <5 mm, V6 were considered Q wave equivalents.

## Cardiovascular magnetic resonance studies

A CE-CMR study was performed with a Philips Intera 1.5T scanner (Best, The Netherlands) in all patients. After the usual scout planes had been obtained, steady-state free-precession cine-MR images were acquired in individual long-axis planes and in multiple 10 mm thick short axis slices from the atrioven-

tricular ring to the apex of the left ventricle. Sixteen phases of the cardiac cycle were acquired for each slice and displayed as a loop.

Intravenous gadobutrol (Gadovist,<sup>®</sup> Schering AG, Berlin, Germany) was injected at a dose of 0.1 mmol/kg. A 3D inversionrecovery segmented gradient echo sequence was acquired 10 minutes after contrast administration to assess delayed contrast myocardial hyperenhancement (HE). Inversion times were adjusted to null the signal from normal myocardium (200–300 ms). This sequence was prescribed in multiple short axis planes using the same orientation as the cine-MR images and acquired during a patient breath-hold of approximately 20 seconds.

MI was considered transmural if at least 1 segment of the North America Society of Imaging (NASI) statement (27) fulfilled the CMR criteria for being transmural (HE in > 50% of wall thickness). To correlate CMR and ECG findings, we divided the left ventricle into two zones (Fig. 1): anteroseptal with some lateral and usually also inferior involvement, perfused by the left anterior descending artery (LAD) and its branches, and inferolateral perfused by the right coronary artery (RCA) and left circumflex artery (LCX) and its branches. Each zone was then divided into different areas and segments (Fig. 1) according to the NASI statement (27). Ce-CMR areas were identified by 3 experts in imaging, and its correspondence with the myocardial NASI segments was determined. According to the presumed site of coronary occlusion, four areas of infarction





were defined in the anteroseptal zone affecting: 1) septum and part of the anterior wall (A1); 2) apical area with or without extension to upper but not basal part of the anterior and septal walls (A2); 3) extensive involvement of the anterior and septal wall also with lateral involvement (A3); and 4) limited part of the anterior wall, especially the middle segment often with some extension to the middle/low lateral wall (A4). Three areas of infarction were defined in the inferolateral zone affecting: 1) lateral wall (B1); 2) inferior wall (B2)\* and 3) inferolateral wall (B3). The bull's eye distribution of these areas with all the segments involved according to the statement of NASI (27) are shown in Table 1. The seven identified areas were correlated with ECG recordings with the aim of establishing seven ECG patterns that better matched each prespecified area of infarction.

#### **Statistics**

Q-wave MI and CE-CMR location were described by means of percentage. Categorical variables were presented as proportions. To define the ECG patterns that better matched with the seven predefined infarction areas detected by CE-CMR, we calculated the sensitivities and specificities of different ECG patterns by using  $2 \times 2$  contingence tables. In each of these tables, the presence or absence of CE-CMR location was correlated with the presence or absence of Q-waves or equivalents in different leads. Finally, we defined seven ECG patterns that presented the highest sensitivity and specificity and global concordance that was calculated by using the Kappa index.

#### RESULTS

## Q/non-Q-wave MI

Forty of 48 patients presented Q-wave MI or equivalents in chronic phase. The ECG in the other 8 cases could be considered normal or borderline (2 with positive T wave in V1 and 1 with slurrings at the end of QRS) (see Table 1).

# Correspondence between CMR/ECG for MI location

MI were grouped according to the 7 predefined areas of infarction as shown by CE-CMR and 7 ECG patterns that better matched with these areas. Table 1 shows this relationship with the corresponding ECG patterns sensitivity and specificity and the name given to the infarction. The global concordance between CE-CMR and ECG was 75%. Some examples of CMR images and their corresponding ECG patterns are displayed in Figs. 2–5. Three cases with limited anterolateral infarction and 5 cases of MI of the inferolateral wall infarctions presented normal ECG in the chronic phase.

#### DISCUSSION

In this study, we present 7 ECG patterns that may be useful to locate myocardial necrosis as assessed by CE-CMR.

O-wave MI location based on correlation of pathologic ECG studies has several limitations owing to technical aspects, and, as a consequence, these correlations are being performed only in few cases, usually in patients with extensive MI. The correspondence between ECG findings and recent imaging techniques has become crucial to overcome these limitations. Echocardiography is the easiest comparative diagnostic imaging technique (17, 18, 28, 29). However, echocardiography frequently overestimates the infarcted area; therefore, its reliability is not excellent. Scintigraphy is very reliable to detect ischemic (18) and infarcted areas (30), but it has been demonstrated that the degree or even the presence of some types of necrosis (non-Q-wave infarction) may be underestimated (31). Recently, CE-CMR after gadolinium injection has permitted an accurate identification of necrotic areas when compared to pathologic studies (32). Furthermore, in clinical trials CE-CMR has been shown to be extremely reliable in assessing not only the extension of the infarcted area but also its transmurality (20-21, 24). Thus, CE-CMR has become the "gold standard" method for quantifying necrotic myocardial mass and also for differentiating between transmural and nontransmural infarcted areas in the chronic phase of ischemic heart disease (20).

To our knowledge, the correlation between CE-CMR infarcted area and Q-wave has only been performed partially and with a small number of patients (23, 24). These studies focus mainly on detection and differentiation between transmural and non-transmural myocardial infarction, and its correspondence to the appearence of Q-wave in the surface ECG considered as an equivalent of transmural compromise. No attention was paid to the correlation between exact localization of MI based on the leads where Q-waves were observed and CE-CMR findings. However, such a correlation is useful not only for academics but also for everyday clinical practice. Ascertaining the area involved through the ECG pattern has important clinical implications and adds more information to quantification of the infarcted myocardial mass.

We agree with the previously presented consensus of the North American Imaging Techniques Societies (27) that divided the heart into 17 segments corresponding to the four heart walls (anterior, septal, lateral, and inferior). However, we believe that the decision of this consensus to give the name inferior wall to what is really inferoposterior wall must be discussed. Segment 4 is named differently by electrocardiologists (posterior) or imaging experts (inferobasal). As may be demonstrated by CE-CMR studies, the basal part of this wall sometimes arches more upwards and therefore converts into an authentic posterior position. Furthermore, this posterior part may be smaller or larger according to the position of the heart in the thorax. In very vertical hearts, it may be almost exclusively posterior. According

<sup>\*</sup>Inferior wall corresponds to segments 4,10 and 15 according to the statement of NASI (Cerqueira 2002). Usually, ECG books refer to inferoposterior wall, with segment 4 being named posterior and in the NASI statement inferobasal (see Discussion). In this article, the term inferior wall will be used instead of inferoposterior, and inferobasal segment instead of posterior segment ( $n^{\circ}$  4).

**Table 1.** Correlations between the different myocardial infarction (MI) types with its infarction area assessed by CMR, ECG pattern and name given to the infraction. The grey zone and arrows seen in bull's eye correspond to infarction areas and its possible extension. Two cases of A-4; three of B-1; one of B-2 and two of B-3 presented normal ECG

Type of MI		Infarction area (CMR)	ECG pattern	Name given to MI
ANTEROSEPTAL ZONE	<b>A1</b> n = 7	1 2 8 13 12 6 12 12 12 12 12 12 13 12 12 13 12 12 13 12 14 17 16 15 10 10 10 10 10 10 10 10 10 10	Q in V1-2 SE: 86% ES: 98%	Septal
	<b>A2</b> n = 7	1 2 8 13 12 6 14 17 16 3 9 15 11 4	Q in V1-2 to V4-V6 SE: 86% ES: 98%	Apical/ anteroseptal
	<b>A3</b> n=5	$\begin{array}{c} & 1 \\ & 1 \\ & 2 \\ & 4 \\ & 4 \\ & 14 \\ & 17 \\ & 16 \\ & 3 \\ & 9 \\ & 15 \\ & 11 \\ & 5 \\ & 4 \\ & 4 \\ \end{array}$	Q in V1-2 to V4-V6 I and VL SE: 80% ES: 98%	Extensive anterior
	<b>A4</b> n = 5	2 8 14 17 16 3 9 15 11 5 4	Q(qs or qr) in VL (I) and sometimes V2-3 SE: 40% ES: 100%	Limited anterolateral
INFEROLATERAL ZONE	<b>B1</b> n=6	1 7 8 13 9 15 12 6 12 6 12 6 13 9 15 12 6 14 12 6 14 15 12 6 14 15 12 12 12 12 12 12 12 12 12 12	Q (qr or r) in I, VL, V5-6 and/or RS in V1 SE: 50% ES: 98%	Lateral
	B2 n = 8	1 2 8 13 13 12 6 3 9 15 11 5 4 4 4	Q in II, III, VF SE: 87,5% ES: 100%	Inferior
	<b>B3</b> n = 10	1 2 8 13 12 6 12 6 12 6 12 6 13 12 6 13 12 6 13 12 6 13 12 6 13 12 6 13 12 6 13 12 6 13 12 6 13 12 6 13 13 15 15 15 15 15 15 15 15 15 15	Q in II, III, VF (B2) + Q in I,VL,V5,6 and /or RS in V1(B1) SE: 80% ES: 100%	Inferolateral

to terminology used in electrocardiography for over 60 years, the term posterior infarction, and consequently also posterior ischemia and injury, is currently used to locate the infarcted area in the basal part of the inferoposterior wall (mainly segment 4), usually due to occlusion of the non-dominant LCx artery. Nevertheless, it is true that infarction of the basal part (segment 4) of the inferoposterior wall (inferior according Cerqueira's statement; 27) does not present RS in V1 and really this morphology is found in infarction of the inferior part of the lateral wall (segments 5 and 11). Indeed, our results confirm the findings of other authors (21) who already found that lateral infarction originates positive R waves in V1. Thus, R (RS) in V1 does not corresponds to segment 4 because it is really due to lateral infarction (necrosis of segment 5 and 11) and not to necrosis of segment 4.





We consider that segment 4 may be named as suggested by Cerqueira (27) inferobasal instead of posterior, and that positive R wave in V1 is not a criterion of posterior MI but of lateral MI.

It has been considered that the ECG abnormalities observed in II, III, VF leads (ST elevation, negative T wave or Q wave of necrosis) without RS in V1 correspond to involvement of the inferior wall. However, frequently there is involvement not only of inferior wall (segments 4,10,15) but also of the inferior part of the septum (segments 3, 9, and 14). In these cases, the occluded artery is usually the RCA. When the ECG presents Q in II, III



ECG pattern seen in some cases of lateral necrosis. Horizontal long-axis view (A): myocardial HE (arrows) shows non-transmural necrosis of the lateral wall at the basel and mid level. Short axis images at the basal (B) and mid level (C): myocardial HE (arrows) indicates non-transmural necrosis of the lateral wall. At the apical level (D) no myocardial HE is observed.

and VF and RS in V1, the involved area is the inferior wall and inferior part of the lateral wall with or without involvement of the inferior part of the septum.

The limited anterolateral MI (A-4 type) must be differentiated from the lateral MI (B-1) owing to the frequent presence in both of Q wave in I and VL and from septal MI (A-1) owing to the possible presence of q in V2-V3 in both. The presence of low QR not QS in VL often with QR pattern or low R in V5-6 favors lateral MI (B-1), and the presence of QS or QrS in V1 and the lack of pathologic Q wave in VL favors septal MI (A-1).

It has been demonstrated thanks to CE-CMR (22, 24), echocardiographic, angiographic, or scintigraphic (14–17) correlations, that QS morphology observed in VL (and sometimes in I) is due to infarction produced by LAD subocclusion involving diagonal arteries. In these cases, the necrosis is located in the mid-anterior wall often with certain propagation to the midlow anterior part of the lateral wall. This may be explained by the fact that this area is perfused by the first diagonal artery, while the inferior part and the majority of the basal-anterior part

of the lateral wall is perfused by the LCX (OM) or intermediate artery. Therefore, the term "high lateral infarction" applied to QS in VL (and sometimes in I lead) is confusing since the above-mentioned morphology appears in the absence of necrosis of the basal area of this wall. On the other hand, when the infarction involves mainly the lateral wall (occlusion of the obtuse marginal artery), "QR" is more frequently seen than "QS" morphology and is also seen in V5-V6.

According to the classical concept, the QS pattern recorded from V1 to V4 was considered as anteroseptal infarction, while contemporary knowledge demonstrates that it represents apical involvement (19). However, it is clear that many cases of isolated apical infarction do not present QS in V1 to V4 (33). Therefore, this criterion is highly specific for apical involvement but relatively insensitive. In our study we included cases of apical infarction with or without anterospetal extension in one group (A-2, named apical/anteroseptal) as in these cases we have not found any differences in the ECG pattern in precordial leads. This is partially due to the fact that the first vector of depolarization,



T. Note that despite clear inferobasal necrosis (gadolinium HE in A), V1 shows rS pattern ant not RS (as seen in some cases of lateral infarction). Images of the short-axis view shows myocardial HE (arrows) at the basal (A) and mid (B) levels corresponding to transmural necrosis of the inferior wall at the basel and mid level. No myocardial HE is observed at the apical level indicating absence of myocardial necrosis(C).

responsible for R in V1-V3 is originated in the mid-low part of septum (13). Probably, in case of isolated apical involvement, it is more frequent to find Q-waves in inferior leads. Therefore, in our experience QS from V1-2 to V5-6 may be found in apical infarction with and without anteroseptal extension.

We found a good correspondence between the infarcted area detected by CE-CMR and ECG pattern (75% of global concordance), considering that cases with normal ECG were also included. In fact, when we assessed, in other study in process, the accuracy of MI location by the leads containing Q waves of equivalent (from ECG to CE-CMR), we found that the correlation is even better.

Nowadays, due to widely used reperfusion techniques, especially in case of thrombolytic treatment, it is impossible to be sure about the place of occlusion in the culprit artery in the acute phase of myocardial infarction while assessing the angiographic findings in the chronic phase of MI. Nevertheless, it can be deducted basing on the already performed correlations between ECG and angiographic findings in the acute phase (10). In this work we did not focus on determination of the culprit artery or the place of its occlusion. Attention was paid to necrosis as the final consequence of coronary occlusion assessed by CE-CMR and its electrocardiographic expression.

## LIMITATIONS

Although these results are very encouraging regarding the correspondence between CE-CMR technique as the gold standard and ECG for MI location, we must acknowledge the limitations, owing to the reduced number of cases and the fact that only cases with ACS and ST elevation in the acute phase of MI were included. Hence, a larger prospective study is required for a definitive statement based on these correlations to be made.

#### CONCLUSIONS

1. Seven pre-specified areas of myocardial infarction detected by CMR technique had good concordance (75%) with 7 ECG patterns (Table 1). 2. Seventeen percent of MI detected by CE-CMR presented normal ECG. 3. Infarction of the lateral wall (especially segments 5 and 11) and not of the inferobasal part of the inferior wall (segment 4) explains RS morphology in V1. 4. Infarction of mid anterior and mid-lateral wall and not of high lateral wall is responsible of QS morphology in VL (I).

#### ACKNOWLEDGEMENT

We would like to thank to Creu Blanca Medical Center, Barcelona.

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