# Original article

# A Prominent R Wave in V<sub>1</sub> but not in V<sub>2</sub> Is a Specific Sign of a Large Lateral Transmural Infarction

Daniele Rovai, a,b,\* Gianluca Di Bella, Giuseppe Rossi, Alessandro Pingitore, a,b and Antonio L'Abbated

- <sup>a</sup> CNR, Istituto di Fisiologia Clinica, Pisa, Italy
- <sup>b</sup> Cardiologia clínica, Fondazione Toscana Gabriele Monasterio, Pisa, Italy
- <sup>c</sup> Dipartimento di Cardiologia, Università di Messina, Messina, Italy
- <sup>d</sup> Instituto di Scienze della Vita, Scuola Superiore Sant'Anna, Pisa, Italy

Article history: Received 6 March 2012 Accepted 28 May 2012 Available online 24 September 2012

Keywords: Electrocardiography Myocardial infarction Magnetic resonance imaging Imaging Coronary disease

Palabras clave: Electrocardiografía Infarto de miocardio Resonancia magnética Exploraciones de imagen Enfermedad coronaria

### ABSTRACT

Introduction and objectives: In the absence of right ventricular hypertrophy or bundle-branch block, a prominent R wave in  $V_1$  or  $V_2$  is considered to reflect a lateral myocardial infarction. We investigated the differences in infarct location, size and transmural extent between patients with prominent R wave in  $V_1$  and those with prominent R wave in  $V_2$ .

*Methods:* We studied 50 patients with a previous first infarction involving left ventricular inferior and/or lateral wall at contrast-enhanced magnetic resonance.

Results: A prominent R wave in  $V_1$  was present in 8 patients (16%), in  $V_2$  in 23 (46%). At magnetic resonance, the infarction involved the inferior wall in 11 patients (22%), the lateral wall in 6 (12%), and both walls in 33 patients (66%). The sensitivity of a prominent R wave in  $V_1$  in detecting a lateral infarction was low (17.9%), while the specificity was high (90.9%). The sensitivity and specificity of a prominent R wave in  $V_2$  were 46.2% and 54.5%, respectively. In patients with a prominent R wave in  $V_1$ , infarct size and lateral and transmural extent were greater than in patients without this pattern (P<.005, <.001, and <.05, respectively); conversely, infarct size and transmural extent in the inferior wall and in its basal-posterior segment were not significantly different. In patients with a prominent R wave in  $V_2$ , infarct size, lateral and transmural extent were not different from patients without this pattern.

Conclusions: Only a prominent R wave in V<sub>1</sub> is a specific sign of large and transmural lateral infarction.

© 2012 Sociedad Española de Cardiología. Published by Elsevier España, S.L. All rights reserved.

# La onda R prominente en $V_1$ pero no en $V_2$ es un signo específico de infarto transmural lateral grande

# RESUMEN

Introducción y objetivos: Si no hay hipertrofia ventricular derecha o bloqueo de rama del haz, se considera que la presencia de una onda R prominente en  $V_1$  o  $V_2$  refleja un infarto de miocardio de la pared lateral. Hemos investigado las diferencias existentes en cuanto a localización, tamaño y extensión transmural del infarto entre los pacientes con una onda R prominente en  $V_1$  y los que presentan una onda R prominente en  $V_2$ .

*Métodos*: Estudiamos a 50 pacientes con un primer infarto previo que había afectado a la pared inferior y/o lateral del ventrículo izquierdo utilizando resonancia magnética con contraste.

Resultados: Se observó la presencia de una onda R prominente en  $V_1$  en 8 pacientes (16%) y en  $V_2$  en 23 pacientes (46%). En las imágenes de resonancia magnética, el infarto afectaba a la pared inferior en 11 pacientes (22%), la pared lateral en 6 (12%) y ambas en 33 (66%). La sensibilidad de la presencia de una onda R prominente en  $V_1$  para la detección de un infarto de cara lateral fue baja (17,9%), mientras que la especificidad fue alta (90,9%). La sensibilidad y la especificidad de una onda R prominente en  $V_2$  fueron del 46,2 y el 54,5% respectivamente. En los pacientes con una onda R prominente en  $V_1$ , el tamaño del infarto y la extensión lateral y transmural fueron mayores que en los pacientes que no mostraban este patrón (p < 0,005, p < 0,001 y p < 0,05 respectivamente); en cambio, el tamaño del infarto y la extensión transmural en la pared inferior y en su segmento posterobasal no mostraron diferencias significativas. En los pacientes con una onda R prominente en  $V_2$ , el tamaño del infarto y la extensión lateral y transmural no fueron diferentes de lo observado en los pacientes sin ese patrón.

Conclusiones: Tan sólo la presencia de una onda R prominente en  $V_1$  constituye un signo específico de un infarto lateral grande y transmural.

© 2012 Sociedad Española de Cardiología. Publicado por Elsevier España, S.L. Todos los derechos reservados.

<sup>\*</sup> Corresponding author: CNR, Istituto di Fisiologia Clinica, Via Moruzzi 1, 56124 Pisa, Italy. E-mail address: drovai@ifc.cnr.it (D. Rovai).

#### **Abbreviations**

ECG: electrocardiogram LV: left ventricular MI: myocardial infarction

MRI: magnetic resonance imaging

### **INTRODUCTION**

For more than 45 years a tall and broad R wave in right precordial leads, in the absence of right ventricular hypertrophy or bundle branch block, has been considered the landmark of posterior myocardial infarction (MI). This criterion is still adopted in current guidelines, and is based on the assumption that the infarction vector of the posterior wall (the basal portion of left ventricular [LV] inferior wall) faces leads  $V_1$  and  $V_2$  and produces a prominent R wave. According to this theory, a tall and broad R wave in right precordial leads is equivalent to a posterior Q-wave.

More recently, a prominent R wave in right precordial leads has been correlated with infarct location in contrast-enhanced cardiac magnetic resonance imaging (MRI). These studies have shown that the infarction vector of the basal segment of the inferior wall faces leads  $V_3$  and  $V_4$  and does not generate a prominent R wave in  $V_1$ . On the contrary, the infarction vector in the case of the lateral wall infarction faces  $V_1$  and often generates a prominent R wave in this lead. To investigate the relationship between the different electrocardiographic patterns of necrosis and MI location, size, and transmural extent, we found that a tall and broad R wave in  $V_1$  or  $V_2$  is a more powerful predictor of lateral MI size than lateral Q waves. However, it is still unclear whether a prominent R wave in  $V_1$  is equivalent to a prominent R wave in  $V_2$  in detecting a lateral MI.

With these considerations in mind, we undertook this study to investigate the differences in MI location, size, and transmural extent between patients with prominent R wave in  $V_1$ , and those with prominent R wave in  $V_2$ .

# **METHODS**

# **Patients**

We studied a group of patients with a previous first Q-wave MI documented by clinical records, using contrast-enhanced MRI and electrocardiogram (ECG). We excluded patients with MI within the last 30 days, patients with multiple MIs or patients with contraindications to the MRI. We also excluded patients with inadequate image quality of the MRI scan or with any condition that could have altered QRS shape and pattern, such as Wolff-Parkinson-White syndrome, left anterior or posterior hemiblock or bundle-branch block, severe LV hypertrophy, severe chronic obstructive pulmonary disease, or hypertrophic or dilated cardiomyopathy. Finally, we excluded patients with evidence of MI involving the anterior, anteroseptal, or apical segments.

Of the 10 828 patients admitted in our Cardiovascular Division between July 2001 and June 2009, we prospectively studied 193 patients with previous Q-wave MI by contrast-enhanced MRI.

From this population, we retrospectively collected a group of patients with previous first Q-wave MI involving the inferior and/ or the lateral wall of the left ventricle. Specifically, of the entire

group of 193 patients, 29 were excluded because of multiple MIs, 27 because of associated conditions that could have altered ORS shape, and 1 patient because of inadequate image quality. Of the remaining 136 patients, 86 were excluded because the MI involved the anterior, anteroseptal, or apical segments at MRI, or because an inferior and/or lateral MI at MRI was even minimally extended to the anterior, anteroseptal, or apical segments. Thus, the study population included 50 patients with areas of delayed contrast enhancement involving one or more of the following segments: basal inferoseptal, basal inferior, basal inferolateral, basal anterolateral, mid inferoseptal, mid inferior, mid inferolateral, mid anterolateral, apical inferior, or apical lateral. The study conforms to the ethical guidelines of the declaration of Helsinki; the institute's committee on human research approved its protocol. All patients signed their informed consent before enrollment in the study. The patient characteristics are shown in Table.

# **Magnetic Resonance Imaging Data Acquisition**

The protocol consisted of cine MRI to evaluate global LV function and contrast-enhanced MRI to determine location, size, and transmural extent of MI. The MRI was performed using a 1.5T whole-body scanner (GE Medical Systems, Milwaukee, WI). A fourelement cardiac phased-array receiver surface coil was used for signal reception. We used a breath-hold, segmented-gradient, fastimaging echo employing a steady-state acquisition ECG-triggered sequence to evaluate global LV function by standard parameters. In each patient a total of 9 to 12 short-axis views (depending on the LV volume) and 2 long-axis views (one vertical and one horizontal) were acquired, with a minimum of 30 cine frames for each slice. From 10 to 15 min after bolus injection of gadoliniumdiethylenetriamine pentaacetic acid (Gadovist, Schering, Berlin, Germany; 0.2 mmol/kg), images were acquired at end-diastole in the same views. A fast-gradient echo inversion recovery sequence was used. The inversion time was optimized until the disappearance of the signal from the viable myocardium.

Characteristics of Patients

| Number                                  | 50        |
|-----------------------------------------|-----------|
| Age, years                              | 65 (11)   |
| Male, %                                 | 90        |
| Family history of CAD, %                | 37        |
| Diabetes mellitus, %                    | 39        |
| Hypercholesterolemia, %                 | 53        |
| Hypertriglyceridemia, %                 | 12        |
| Arterial hypertension, %                | 56        |
| Smoking habit, %                        | 53        |
| Obesity, %                              | 30        |
| No. of stenosed vessels                 | 2 (0.9)   |
| LVEDV, mL/m <sup>2</sup>                | 96 (30)   |
| LVESV, mL/m <sup>2</sup>                | 54 (27)   |
| LVEF, %                                 | 47 (13)   |
| LV mass, g/m <sup>2</sup>               | 81 (22.5) |
| DCE Extent, % of entire LV              | 8 (4.4)   |
| Segments DCE transmural extent 1%-25%   | 1.5 (1.5) |
| Segments DCE transmural extent 26%-50%  | 1.6 (1.6) |
| Segments DCE transmural extent 51%-75%  | 1 (1.2)   |
| Segments DCE transmural extent 76%-100% | 0.6 (1.1) |
|                                         |           |

CAD, coronary artery disease; DCE, delayed contrast enhancement; LV, left ventricular; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESV, left ventricular end-systolic volume.

# **Magnetic Resonance Imaging Data Analysis**

The LV endocardial borders were manually drawn on LV short-axis images corresponding to end-diastolic and end-systolic phases to calculate LV volumes and the ejection fraction. The LV myocardium was divided according to a 17-segment model.<sup>8</sup> To assess location, size, and transmural extent of the MI, in each image the boundaries of contrast-enhanced areas were automatically traced (using a signal intensity cut-off of >5 SD over the average of normal remote myocardium), and manually corrected when needed. The reproducibility of this method has been previously validated.<sup>9</sup>

To measure the extent of the MI, in each image the myocardium was automatically divided into equiangular sectors starting from the anterior septal insertion. Each sector was subdivided into 100 radiants, and the extent of contrast enhancement in each radiant was measured automatically (Fig. 1). Three to four contiguous slices were averaged to obtain the extent of the MI in each segment. The transmural extent of the MI in each segment was scored according to a 4-point scale, where 1 corresponds to < 25% of LV myocardial thickness, 2 is between 26% and 50%, 3 between 51% and 75%, and 4 corresponds to an extent>75% of LV thickness. The infarct size was calculated as the sum of the scores of all 10 segments of the inferior and lateral walls. Infarct extent in the inferior or lateral wall was calculated as the sum of the scores in the 5 inferior or 5 lateral segments. Infarct transmural extent was calculated as the mean score in the segments with contrast enhanced myocardium.

# Electrocardiogram

A 12-lead ECG was recorded at a speed of 25 mm/s and a voltage of 10 mm/mV. Two cardiologists, unaware of the MRI data, analyzed the ECG tracings off-line. In the case of disagreement regarding the ECG interpretation, a consensus was reached by reading the tracing together. The criteria for identification of the ECG patterns of necrosis were defined before the analysis. Q waves were considered pathological if they were >0.04 s in duration. The R wave in  $V_1$  or  $V_2$  was considered prominent if the R/S ratio was>1 and the R wave duration was>0.04 s.  $^{3,10,11}$  The patients were categorized on the basis of a prominent R wave in  $V_1$  or a prominent R wave in  $V_2$ .

# **Statistical Analysis**

Quantitative data are expressed as mean $\pm 1$  SD, qualitative data as percentage. The ability of the different ECG patterns of necrosis to detect MI location was evaluated as sensitivity and specificity, according to their definition. The difference between MI size,

horizontal extent, and transmural extent in patients with the different ECG patterns of necrosis was tested by the analysis of variance. All statistical tests were 2-tailed; a *P*-value<.05 was considered significant. Statistical analysis was performed with the JMP 9 software (SAS Institute Inc.).

# **RESULTS**

The 12-lead ECG showed a prominent R wave in  $V_1$  in 8 patients (16%); all these patients also had a prominent R wave in  $V_2$ . The ECG showed a prominent R wave in  $V_2$  but not in  $V_1$  in 15 patients (30%). Thus, 23 patients (46%) showed a prominent R wave in  $V_2$ . Inferior Q waves of necrosis were present in 27 patients (54%). These Q waves were associated with a prominent R wave in  $V_1$  in 5/8 patients, with a prominent R wave only in  $V_2$  in 10/15 patients, and were not associated with a prominent R wave in 12 patients. At cardiac MRI, the infarction scar was located only in the inferior wall in 11 patients (22%), only in the lateral wall in 6 patients (12%), and in both inferior and lateral walls in 33 patients (66%).

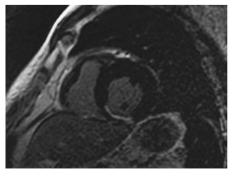
# Prominent R Wave in $V_1$ or $V_2$ and Location of Myocardial Infarction

Of the 8 patients with a prominent R wave in  $V_1$ , 7 showed areas of delayed contrast enhancement in the lateral wall; specifically, the infarction scar was located only in the lateral wall in 2 patients, and in both inferior and lateral walls in 5. In the remaining patient with a prominent R wave in  $V_1$ , a small subendocardial area of delayed contrast enhancement was located in the mid-inferior segment. Of the 42 patients without prominent R wave in  $V_1$ , 32 showed areas of necrosis in the lateral wall, and 10 did not. Thus, the sensitivity of a prominent R wave in  $V_1$  in detecting a lateral MI was low (17.9%), while the specificity was high (90.9%). The positive and negative predictive values were 87.5% and 23.8%, respectively.

Of the 23 patients with prominent R wave in  $V_2$ , 18 showed lateral infarction scars, and 5 did not. Of the 27 patients without this pattern, 21 showed evidence of lateral necrosis at MRI, and 6 did not. Thus, the sensitivity and the specificity of a prominent R wave in  $V_2$  in detecting lateral infarctions were 46.2% and 54.5%, respectively.

# Prominent R Wave in V<sub>1</sub>, Infarct Size, and Transmural Extent

In patients who presented a prominent R wave in  $V_1$ , the overall size of the infarction at contrast-enhanced MRI, the extent of the MI in the lateral wall, and infarct transmural extent were higher than in patients without this electrocardiographic pattern (Fig. 2).



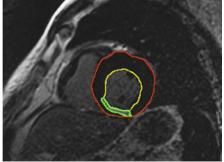


Figure 1. Contrast-enhanced, short-axis image of the heart. The lines in the right panel correspond to left ventricular endocardial (yellow) and epicardial boundaries (red), and to the edges of contrast- enhanced area (green).

Conversely, infarct size in the inferior wall and in its basal segment (formerly called posterior) were not significantly different between patients with and without a prominent R wave in  $V_1$  (Fig. 3).

# Prominent R Wave in V<sub>2</sub>, Infarct Size, and Transmural Extent

In patients with prominent R wave in  $V_2$ , the overall infarct size and its lateral and transmural extent were not significantly different from those of patients without such a pattern (Fig. 4), nor were the size of the MI in the inferior wall (P=.348) or in its inferobasal segment (P=.187).

In the 20 patients with inferior Q waves, infarct size in the lateral wall was not significantly different (P=.419) from that of patients without Q waves, while MI extent in the inferior wall was significantly higher (P=.017).

### DISCUSSION

This study shows that a prominent R wave in  $V_1$  and in  $V_2$  are markers of a lateral MI. However, a prominent R wave in  $V_1$  is a specific marker but not very sensitive, while a prominent R wave in  $V_2$  is a more sensitive but less specific marker. In practical terms, a prominent R wave in  $V_1$ —in the absence of right ventricular hypertrophy or bundle branch block—is a diagnostic sign of a lateral infarction, while a prominent R wave in  $V_2$  may lead to misdiagnosis. Although these conclusions can be envisaged by previous studies, this is the first investigation that compares the different meanings of a prominent R wave in  $V_1$  and  $V_2$ .

The relationship between an R wave in  $V_1$  and  $V_2$  and infarct location has been investigated in several studies. In a previous study we evaluated patients with Q-wave MI or equivalents. Thus, we selected patients based on electrocardiographic evidence of necrosis, while in this study we selected patients based on the MRI evidence of infarction. Furthermore, we previously included all possible infarct locations, while in the present study we selected only patients with inferior, lateral, or inferolateral MI. This study is also different from the previous ones performed by Bayes de Luna et al.,  $^{3-6}$  where the MRI data were categorized in a binary way (as presence or absence of a previous MI) while we have included a quantitative data analysis on infarct size and transmural extent.

According to the results of various studies, including the present one, the commonly utilized nomenclature of MI location<sup>2</sup>

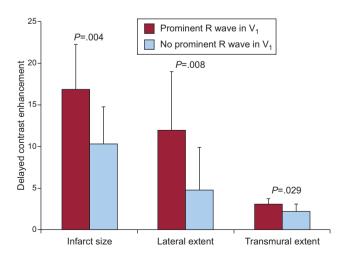
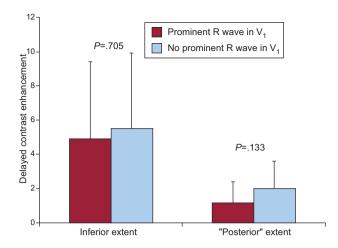


Figure 2. Myocardial infarct size, lateral and transmural extent in patients with and without a prominent R wave in  $V_1$  (in red and blue).



**Figure 3.** Myocardial extent in LV inferior wall and in its inferobasal segment in patients with and without a prominent R wave in  $V_1$  (in red and blue, respectively).

should be reconsidered. Specifically, a prominent R wave in right precordial leads should be considered a sign of lateral MI, not of posterior MI. This is not a matter of semantics, since the LV lateral wall is generally perfused by the left circumflex coronary artery, often by the obtuse marginal branch, while LV inferobasal wall is usually perfused by the right coronary artery. This consideration should be taken into account when patients undergo coronary arteriography and myocardial revascularization. Finally, although this was not the end-point of the study, our results confirm that the Q-waves of necrosis in the inferior leads not only have no relationship with lateral MI, but correspond to a scar located in the inferior LV wall.

This study revealed the innovative information that a prominent R wave in  $V_1$  carries quantitative information on MI size and transmural extent. In fact, a prominent R wave was associated with a larger infarct size, while in the absence of this sigh the infarction was smaller and more confined to the subendocardial layers. In a study of 100 patients with previous MI, Moon et al. found the Q waves to be indicative of a large infarct; in addition, a strong relationship was found between classification as a Q-wave MI and quintiles of transmural extent of MI measured by contrastenhanced MRI. In another study, although several parameters were predictors of Q waves at univariate analysis, multivariate analysis showed that quantified scar tissue extent was the single

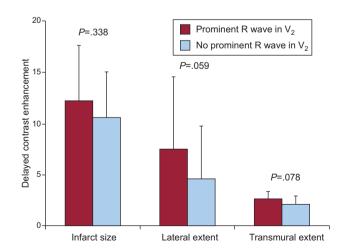


Figure 4. Myocardial infarct size, lateral and transmural extent in patients with and without a prominent R wave in  $V_2$  (alone or associated with a prominent R wave in  $V_1$ ) (in red and blue).

best predictor of Q waves on the ECG.  $^{13}$  In line with the above evidence, the results of the present study allow us to conclude that a large lateral MI, which generates a big infarction vector, is needed to produce a prominent R wave in  $V_1$ . Accordingly, the "falsenegative" results of the patients with lateral infarction without prominent R waves could be explained by the fact that their infarction was smaller and limited to the subendocardial layers, in other words not large enough to generate a prominent infarction vector.

In previous studies<sup>3–6</sup> a prominent R wave in V<sub>1</sub> had a specificity of 100% in detecting a lateral infarction. In our study one patient with prominent R wave in V<sub>1</sub> did not show a lateral infarction, leading to a specificity of 90.9%. However, the duration of the R wave in V<sub>1</sub> in our patient was<0.04 s, while by definition a prominent R wave has to be wide, with a duration >0.04 s.<sup>3,10,11</sup> Furthermore, although an acute coronary syndrome was excluded, the T waves were negative (max. 0.25 mV) from V<sub>1</sub> to V<sub>3</sub>. If this patient was excluded from the study, our specificity would have been 100% as well.

This study is affected by several limitations. Because of the extreme variability in coronary anatomy among individual patients,14 the considerations made regarding the coronary arteries supplying the different LV walls are only true in median terms. In previous studies, various authors have investigated several parameters that describe a prominent R wave, as the R/S ratio or R wave duration; these parameters were not analyzed in the present study. Furthermore, the sample size in this study is limited, and should be expanded in larger cooperative studies. Although very specific, a prominent R wave in V<sub>1</sub> was not a very sensitive sign of lateral infarction, thus limiting its utilization for diagnostic purposes. Finally, it should be kept in mind that the relationships between a prominent R wave in V<sub>1</sub> or V<sub>2</sub> and infarction scars found in patients with previous MI cannot be extrapolated to the acute phase of the disease or to ST segment shifts.

# **CONCLUSIONS**

A pathologic R wave in  $V_1$  is a specific marker of lateral infarction, and unmasks a large infarction scar, transmurally well extended. A prominent R wave in  $V_2$  is a more sensitive but less specific marker of lateral MI, and can induce a reasonable suspicion.

# **FUNDING**

This study was supported by institutional grants of the National Research Council (CNR), Italy.

#### CONFLICTS OF INTEREST

None declared.

#### REFERENCES

- Perloff J. The recognition of strictly posterior myocardial infarction by conventional scalar electrocardiography. Circulation. 1964;30:706–18.
- 2. Wagner GS, Macfarlane P, Wellens H, Josephson M, Gorgels A, Mirvis DM, et al. American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society. AHA/ACCF/HRS recommendations for the standardior; and interpretation of the electrocardiogram: part VI: acute ischemia/infarction: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol. 2009;53:1003-11.
- Bayés de Luna A, Cino JM, Pujadas S, Cygankiewicz I, Carreras F, et al. Concordance of electrocardiographic patterns and healed myocardial infarction location detected by cardiovascular magnetic resonance. Am J Cardiol. 2006;97:443–51.
- Cino J, Pujadas S, Carreras F, Cygankiewicz I, Leta R, Noguero M, et al. Utility of contrast- enhanced cardiovascular magnetic resonance to assess the sensitivity and specificity of different ECG patterns to locate Q-wave myocardial infarction. I Cardiovasc Magn Reson. 2006;8:1–10.
- 5. Bayés de Luna A, Wagner G, Birnbaum Y, Nikus K, Fiol M, Gorgels A, et al. A new terminology for left ventricular walls and location of myocardial infarcts that present Q wave based on the standard of cardiac magnetic resonance imaging: a statement for healthcare professionals from a committee appointed by the International Society for Holter and Noninvasive Electrocardiography. Circulation. 2006;114:1755–60.
- Bayés de Luna A, Cino J, Goldwasser D, Kotzeva A, Elosua R, Carreras F, et al. New electrocardiographic diagnostic criteria for the pathologic R waves in leads V1 and V2 of anatomically lateral myocardial infarction. J Electrocardiol. 2008;41:413–8.
- 7. Rovai D, Di Bella G, Rossi G, Lombardi M, Aquaro GD, L'Abbate A, et al. Q-wave prediction of myocardial infarct location, size and transmural extent at magnetic resonance imaging. Coron Artery Dis. 2007;18:381–9.
- Cerqueira MD, Weissman NJ, Dilsizian V, Jacobs AK, Kaul S, Laskey WK, et al. American Heart Association Writing Group on Myocardial Segmentation and Registration for Cardiac Imaging, Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: a statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. Circulation. 2002:105:539–42.
- Positano V, Pingitore A, Giorgetti A, Favilli B, Santarelli MF, Landini L, et al. A fast and effective method to assess myocardial necrosis by means of contrast magnetic resonance imaging. J Cardiovasc Magn Reson. 2005;7:487–94.
- Bough EW, Boden WE, Korr KS, Gandsman EJ. Left ventricular asynergy in electrocardiographically 'posterior' myocardial infarction. J Am Coll Cardiol. 1984:4:209-15
- Nestico PF, Hakki AH, Iskandrian AS, Anderson GJ. Electrocardiographic diagnosis of posterior myocardial infarction revisited: a new approach using a multivariate discriminant analysis and thallium-201 myocardial scintigraphy. I Electrocardiol. 1986:19:33-40.
- 12. Moon JC, De Arenaza DP, Elkington AG, Taneja AK, John AS, Wang D, et al. The pathologic basis of Q-wave and non-Q-wave myocardial infarction: a cardio-vascular magnetic resonance study. J Am Coll Cardiol. 2004;44:554–60.
- Kaandorp TA, Bax JJ, Lamb HJ, Viergever EP, Boersma E, Poldermans D, et al. Which parameters on magnetic resonance imaging determine Q waves on the electrocardiogram? Am J Cardiol. 2005;95:925–9.
- Pereztol-Valdes O, Candell-Riera J, Santana-Boado C, Angel J, Aguade-Bruix S, Castell-Conesa J, et al. Correspondence between left ventricular 17 myocardial segments and coronary arteries. Eur Heart J. 2005;26:2637–43.