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Introduction of Guest Editorial

In this issue of the *Journal*, Cino Juan Manuel, MD, Guillem Pons Llado, and Antonio Bayés de Luna and their colleagues have used CMR to demonstrate the reliability of the electrocardiogram for defining the location of myocardial infarction. The following editorial was invited in view of the importance of their manuscript.

Gerald M. Pohost, MD
Editor-in-Chief

GUEST EDITORIAL

A Reappraisal of Infarct ECG Patterns Based on CMR

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Accurate detection of myocardial infarction (MI) in its chronic state is a clinically-significant issue. It has been demonstrated, from the epidemiological point of view, that the mortality rate in individuals who have suffered an MI in the past, which can be silent in up to 15%, is higher than that of the general population (1).

Electrocardiogram (ECG) remains as the most popular and simplest technique for detecting a chronic MI since pathologic Q-waves or equivalents (R or RS waves in lead V1, low-amplitude R wave in lateral leads) remains a very specific, although relatively insensitive criteria for MI diagnosis and location (2). ECG recordings, however, can be misleading in some cases of previous MI, particularly when Q-waves are absent and tracings obtained during the acute phase are not available (3). On the other hand, evidence that particular ECG abnormalities, such as RS or morphology in lead V1 or qR in inferior leads, are due to MI is dubious.

Non-invasive imaging techniques may detect the presence of a MI, but most also have limitations. Thus, echocardiography may lead to regions with stunned or hibernated, but viable myocardium being considered affected by an MI. On the other hand, the presence of a non-transmural MI may go undetected (4), which may also occur in the case of scintigraphy (5); even regional metabolism by positron emission tomography may be normal in the case of a non-transmural MI (6).

Cardiovascular magnetic resonance (CMR) has recently emerged as a sensitive, specific method for detecting and quantifying MI by means of refined techniques of contrast enhancement (7), with its accuracy in this respect having been proved in pathologic studies in animal models (8). A particularly advantageous feature of CMR is its ability to precisely delineate the extension of a myocardial necrosis (9), an aspect that has been widely admitted, and it can be readily applied as a reference method for comparison of other techniques (5, 6). The value of contrast-enhanced CMR in this field is beyond doubt, since its accuracy in the detection of very small infarcts, such as those caused by alcohol septal ablation for hypertrophic cardiomyopathy (10) or during a coronary angioplasty (11), is well documented. This ability of CMR has afforded further insight into the issue of transmurality of an MI (12), proving, interestingly, that most (99%) patients with a Q-wave MI have some extension of non-transmural necrosis, while 28% of those with a non-Q-wave MI do in fact exhibit transmural necrosis by CMR. Furthermore, 29% of all patients with transmural MI do not present abnormal Q-waves on their ECG. In the opinion of the authors of this study, despite these findings, the classical distinction between Q-wave and non-Q-wave MI retains its clinical value since as the main determinant of the presentation of abnormal Q-waves on ECG is the extension of the necrosis, transmural or not; thus,

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patients with Q-wave MI have larger infarcts than those without Q-waves.

CMR can thus be considered a true gold standard in the detection and location of a myocardial necrosis, a fact that should be regarded as highly beneficial by all cardiologists, particularly those interested in ECG. For decades, pathology studies have been the only reference on which to base anatomical correlations of ECG patterns (13–15). Now, contrast-enhanced CMR is ready to replace autopsy for this purpose, and although some first brilliant studies on CMR-ECG correlations have appeared (12), more information on the correlation between ECG patterns of MI and infarction location has to be gleaned from comparisons with this new reference standard.

In this issue of the *Journal*, a study (16) in which such a comparison was performed is presented. It is based on the concept that up to 7 distinct myocardial areas assessed by CE-CMR exist: 4 in the anteroseptal zone (A-1 to A-4 in Table 1) and 3 in the inferolateral (B-1 to B-3 in Table 1). The global concordance between CE-CMR and ECG was of 75% and 7 ECG patterns have been established. The areas that present more cases with normal ECG are A-4 of the anteroseptal zone and the areas of the inferolateral zone. One important finding of this study was the evidence that infarction that involves mainly the basal part of the inferior wall does not originate RS morphology in V1, in contrast, this morphology is produced by infarction of posterior segments of the lateral wall. Therefore, the name posterior infarction in cases of RS morphology in V1 is incorrect. Furthermore, it has also been demonstrated that QS in VL is not due to necrosis of the high lateral wall but to necrosis of the anterior wall, particularly its middle part.

It is reassuring to observe how the clinical value of the simplest methods, such as ECG, is being increased by findings from the most technologically complex tools such as CMR. Although simple and inexpensive, ECG remains in the front line of diagnostic cardiology nearly one century after its introduction. Of note, Willem Einthoven received the Nobel Prize in Physiology or Medicine in 1924 "for his discovery of the mechanism of the electrocardiogram," while the same honor went in 2003 to Paul Lauterbur and Sir Peter Mansfield "for their discoveries concerning magnetic resonance imaging." Given these common beginnings, and the solid value of CMR proven in its short life, a future parallel to ECG can hopefully be anticipated also for CMR.

REFERENCES

1. Kannel WB, Abbott RD. Incidence and prognosis of unrecognized myocardial infarction: an update on the Framingham study. N Engl J Med 1984;311:1144–7.
2. Bayés de Luna A. Electrocardiographic patterns of ischemia, injury and necrosis. In: Clinical Electrocardiography. 2nd ed. Futura Publishing Company Inc: New York, 1998, pp. 121–161.
3. Bayés de Luna A, Fiol M. Imagen de isquemia, lesíon of necrosis. La electrocardiografia de la cardiopatia isquemica. Correlaciones clinicas e implicaciones pronosticas. Prous Science: Barcelona-Philadelphia. In press.
4. Pandian NG, Skorton DJ, Collins SM, Koyanagi S, Kieso R, Marcus ML, Kerber RE. Myocardial infarct size threshold for two-dimensional echocardiographic detection: sensitivity of systolic wall thickening and endocardial motion abnormalities in small versus large infarcts. Am J Cardiol 1985;55:551–5.
5. Wagner A, Mahrholdt H, Holly TA, Elliott MD, Regenfus M, Parker M, Klocke FJ, Bonow RO, Kim RJ, Judd RM. Contrast-enhanced MRI and routine single photon emission computed tomography (SPECT) perfusion imaging for detection of subendocardial myocardial infarcts: an imaging study. Lancet 2003;361:374–9.
6. Klein C, Nekolla SG, Bengel FM, Momose M, Sammer A, Haas F, Schnackenburg B, Delius W, Mudra H, Wolfram D, Schwaiger M. Assessment of myocardial viability with contrast-enhanced magnetic resonance imaging: comparison with positron emission tomography. Circulation 2002;105:162–7.
7. Simonetti OP, Kim RJ, Fieno DS, Hillenbrand HB, Wu E, Bundy JM, Finn JP, Judd RM. An improved MR imaging technique for the visualization of myocardial infarction. Radiology 2001;218:215–23.
8. Fieno DS, Kim RJ, Chen EL, Lomasney JW, Klocke FJ, Judd RM. Contrast-enhanced magnetic resonance imaging of myocardium at risk: distinction between reversible and irreversible injury throughout infarct healing. J Am Coll Cardiol 2000;36:1985–91.
9. Wu E, Judd RM, Vargas JD, Klocke FJ, Bonow RO, Kim RJ. Visualization of presence, location, and transmural extent of healed Q-wave and non-Q-wave myocardial infarction. Lancet 2001;357:21–8.
10. van Dockum WG, ten Cate FJ, ten Berg JM, Beek AM, Twisk JW, Vos J, Hofman MB, Visser CA, van Rossum AC. Myocardial infarction after percutaneous transluminal septal myocardial ablation in hypertrophic obstructive cardiomyopathy: evaluation by contrast-enhanced magnetic resonance imaging. J Am Coll Cardiol 2004;43:27–34.
11. Ricciardi MJ, Wu E, Davidson CJ, Choi KM, Klocke FJ, Bonow RO, Judd RM, Kim RJ. Visualization of discrete microinfarction after percutaneous coronary intervention associated with mild creatine kynase-MB elevation. Circulation 2001;103:2780–83.
12. Moon JC, De Arenaza DP, Elkington AG, Taneja AK, John AS, Wang D, Janardhanan R, Senior R, Lahiri A, Poole-Wilson PA, Pennell DJ. The pathologic basis of Q-wave and non-Q-wave myocardial infarction. A Cardiovascular Magnetic Resonance Study. J Am Coll Cardiol 2004;44:554–60.
13. Myers GB, Klein HA, Hiratzka T. Correlation of electrocardiographic and pathologic findings in posterolateral infarction. Am Heart J 1949;38:837–62.
14. Rodríguez MI, Anselmi A, Sodi-Pallares D. The electrocardiographic diagnosis of septal infarctions. Am Heart J 1953;45:525–44.
15. Savage R, Wagner G, Ideker R, Podolsky S, Hackel D. Correlation of postmortem anatomic findings with electrocardiographic changes in patients with myocardial infarction. Circulation 1977;55:279–85.
16. Cino JM, Pujadas S, Carreras F, Cygan Kiewicz I, Leta R, Noguero M, Garcia-Moll X, Bayés-Genis A, Pons-Lladó G, Bayés de Luna A. Utility of contrast-enhanced cardiovascular magnetic resonance (E-CMR) to assess how likely is an infarct to produce a typical ECG pattern. J Cardiovasc Magn Reson 2006;8:335–343.