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Myocardial Mapping With Cardiac Magnetic Resonance: The Diagnostic Value of Novel Sequences

Javier Sanz,[a,b,*] Gina LaRocca,[a] and Jesús G. Mirelis[b,c]

[a] The Zena and Michael A. Wiener Cardiovascular Institute and Marie-Josee and Henry R. Kravis Center for Cardiovascular Health, Mount Sinai School of Medicine, New York, United States
[b] Centro Nacional de Investigaciones Cardiovasculares (CNIC), Madrid, Spain
[c] Servicio de Cardiología, Hospital Universitario Puerta de Hierro, Majadahonda, Madrid, Spain

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ABSTRACT

Cardiac magnetic resonance has evolved into a crucial modality for the evaluation of cardiomyopathy due to its ability to characterize myocardial structure and function. In the last few years, interest has increased in the potential of "mapping" techniques that provide direct and objective quantification of myocardial properties such as T_1, T_2, and T_2^* times. These approaches enable the detection of abnormalities that affect the myocardium in a diffuse fashion and/or may be too subtle for visual recognition. This article reviews the current state of myocardial T_1 and T_2-mapping in both health and disease.

Mapeo miocárdico con resonancia magnética cardiaca: valor diagnóstico de las nuevas secuencias

Palabras clave:
Resonancia magnética cardiaca
Mapeo
Miocardiopatía

RESUMEN

La resonancia magnética cardiaca ha evolucionado hasta convertirse en una modalidad diagnóstica esencial en la evaluación de la miocardiopatía, gracias a su capacidad para caracterizar la estructura y la función del miocardio. En los últimos años ha aumentado el interés en el potencial de las técnicas de mapeo que aportan una cuantificación directa y objetiva de las propiedades del miocardio, como los tiempos T_1, T_2 y T_2^*. Estos métodos permiten detectar anomalías que afectan al miocardio de manera difusa o son demasiado sutiles para identificarlas en un examen visual. En este artículo se revisa el estado actual del mapeo miocárdico T_1 y T_2 tanto en salud como en enfermedad.

INTRODUCTION

Standard approaches for myocardial characterization with cardiac magnetic resonance (CMR) include T_1-weighted, T_2-weighted, and late gadolinium enhancement (LGE) imaging that allow the visualization of fatty infiltration, edema, or necrosis/scarring.[1] These sequences rely on relative changes in signal intensity between abnormal and normal myocardium. However, they are hampered by their often semiquantitative nature and their inherent limitations in depicting diffuse myocardial processes with no "normal" reference myocardium at the time of imaging. Myocardial mapping with CMR is quickly evolving as an objective and quantitative approach for the noninvasive characterization of myocardial properties such as extracellular volume expansion, edema, or other abnormalities in tissue composition. In this article, we review state-of-the-art myocardial T_1- and T_2- mapping in health and disease. Older T_2^*-mapping approaches that can detect iron overload or intramyocardial hemorrhage are reviewed elsewhere.[2]

T_1- AND T_2-MAPPING

A detailed description of the physics principles of CMR is beyond the scope of this review. Briefly, CMR generates images by transferring energy to ^{1}H water and fat protons, which is in turn released as they recover their baseline state ("relax"), and which can be detected and mapped into a spatial distribution of protons. The speed of this relaxation is determined by T_1 and T_2 (longitudinal and transverse relaxation times, respectively). T_1 and T_2 times are intrinsic tissue properties that also depend on the magnetic field strength: T_1 lengthens at higher fields whereas T_2 remains relatively constant,[3] although myocardial T_2 tends to shorten.[4] Gadolinium-based contrast agents change relaxation times, specifically shortening T_1.

A T_1- or T_2-map is an image in which signal intensity in each voxel is directly proportional to the T_1 or T_2 time of the tissue within. These times can be compared with those of remote

* Corresponding author: Cardiovascular Institute, Mount Sinai Hospital, One Gustave L Levy Place, Box 1030, New York, NY 10029, United States.
E-mail address: Javier.Sanz@mssm.edu (J. Sanz).

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Abbreviations

CMR: cardiac magnetic resonance
DCM: dilated cardiomyopathy
HCM: hypertrophic cardiomyopathy
LGE: late gadolinium enhancement
LV: left ventricular
MI: myocardial infarction
MOLLI: modified look-locker inversion recovery

myocardium in focal or heterogeneous processes, or with normal reference values in cases of diffuse disease. While LGE detects localized replacement fibrosis,[5] T_1-mapping techniques were initially developed to study diffuse interstitial fibrosis, although their applications continue to expand. The main purpose of T_2-mapping is the detection of edema.[2]

Native T_1 Time

One potential application of T_1-mapping is the quantification of native (or precontrast) myocardial T_1 (Figure 1A). Native T_1 can be prolonged or shortened in a variety of clinical conditions (see below). Since contrast administration is not needed, native T_1-mapping offers diagnostic potential in patients with relative contraindications to gadolinium (ie, advanced renal failure). The histopathological correlates of T_1 remain incompletely elucidated, but T_1 reflects changes in both the intracellular and extracellular compartments, and is influenced by the presence of edema, collagen or other proteins, iron, and lipids.[6]

Postcontrast T_1 Time

T_1 can be calculated after the administration of gadolinium (Figure 1B). Most gadolinium agents are extracellular compounds: they distribute in the intravascular and interstitial compartments but not within the cells. Thus, reduced postcontrast T_1 either reflects access to the intracellular space (loss of cell membrane integrity in, for example, acute necrosis) and/or interstitial space expansion that is largely considered a surrogate of interstitial fibrosis.[7] Although this was the first approach for clinical myocardial T_1-mapping,[8] postcontrast T_1 use has fallen somewhat out of favor because of its dependency on time after gadolinium administration, contrast dose, body composition, renal clearance, heart rate, and hematocrit.[5] Approaches to correct for these variations have nonetheless been proposed.[9–12]

Partition Coefficient (λ)

λ represents the relationship between changes in pre- and postcontrast myocardium and blood T_1 and is calculated as:

$$\lambda = \Delta R1_{myoc} / \Delta R1_{blood}$$

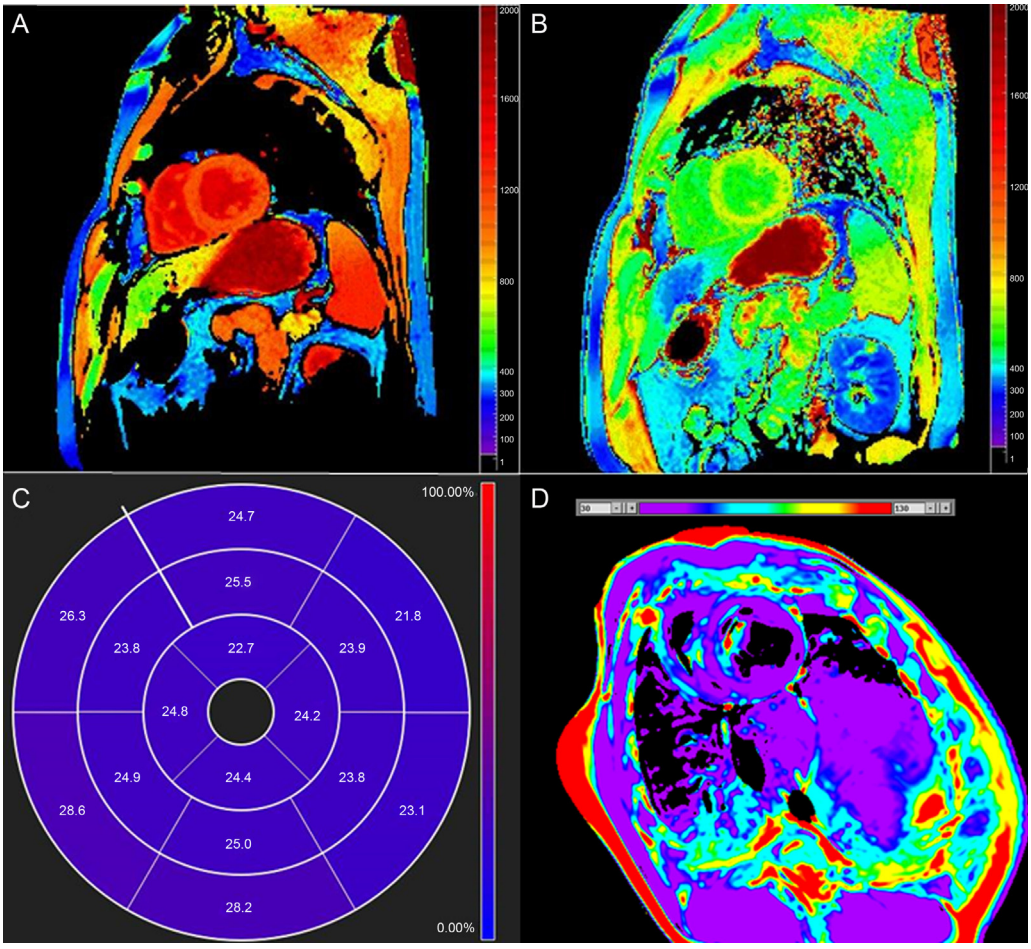

Figure 1. Native myocardial T_1 map (A), postcontrast T_1 map (B), 17-segment extracellular volume diagram (C) and T2 map (D). A-C: normal individual; D: normal experimental animal.