Cardiac Magnetic Resonance T1-rho Mapping at 1.5T

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Introduction

Cardiovascular magnetic resonance (CMR) imaging has evolved as a pivotal modality for the assessment of myocardial pathologies, particularly in the context of myocardial necrosis and focal replacement fibrosis. The cornerstone technique of late gadolinium enhancement (LGE) has proven instrumental in providing valuable insights into cardiac structural abnormalities [1]. However, its limitations in distinguishing between acute and chronic injuries, coupled with a restricted sensitivity to diffuse tissue changes without a healthy myocardial reference, necessitate the exploration of alternative approaches.

Parametric mapping techniques, specifically T1 and T2 mapping, have successfully addressed some of these limitations, enhancing our diagnostic and prognostic capabilities for structural heart diseases. The combination of LGE with multi-parametric mapping has enriched our understanding of cardiac pathologies. Nevertheless, the inherent drawbacks of this combined approach, including prolonged scan times and reliance on gadolinium-based contrast agents, present substantial challenges to health-care costs and CMR availability [2].

Considering these challenges, the quest for a non-contrast CMR technique capable of accurately and quantitatively detecting myocardial injuries of ischemic and non-ischemic etiologies has gained attention. Myocardial T1-rho (T1ρ) mapping, in particular, has emerged as an additional solution to T1 and T2 mapping for characterizing the myocardium without requiring the injection of contrast agent [3, 4]. The fundamental principle underlying T1ρ mapping involves the occurrence of T1ρ relaxation, achieved through spin-locking transverse magnetization with a continuous low power radiofrequency (RF) pulse.

Preliminary in vivo applications of T1ρ mapping have shown promising results, with studies reporting elevated T1ρ values in specific cardiac conditions such as myocardial infarction, hypertrophic and dilated cardiomyopathies, and end-stage renal disease [5–7].

This article will begin with a succinct reminder of the theoretical foundations behind T1ρ mapping. We will then get into the details of how images are collected at 1.5T, and continue with exploring preliminary clinical applications where myocardial T1ρ mapping has proven useful under real-life conditions. Finally, we will discuss the exciting trajectories we might see with this technology in the future.

Theory

Nearly four decades ago, Sepponen et al. [8] delved into one of the initial applications of T1ρ in MRI, although the foundational concept of "spin-locking" dates back to the mid-1950s. While T1 and T2 relaxation times primarily represent inherent physical properties of tissue at a given magnetic field strength, T1ρ stands out by relying not only on tissue properties but also on the characteristics of the applied spin-locking RF pulse – specifically, its amplitude, duration, and module type. The manipulation of these pulse features allows for the modulation of spin-locking and an exploration of how water protons respond to their environment, known as the "lattice".

Siemens Healthineers Disclaimer: This article describes possible future ideas and concepts. It is not intended to describe specific performance and/or safety characteristics of currently planned or future products. Future realization and availability cannot be guaranteed.

Given that spin-locking fields operate within the 2–12 µT range (i.e., the low kHz range), T1ρ relaxation time proves to be sensitive to slow molecular motion processes within the lattice. It therefore offers complementary insights compared to conventional T1 and T2 measurements.

In a T1ρ experiment, the equilibrium magnetization established by the static B_0 magnetic field undergoes a 90° rotation (tip-down) through an RF pulse into the transverse plane (M_{xy}). A spin lock RF pulse with amplitude B_1 is then applied parallel to the magnetization to lock the spins in the rotating frame. Subsequently, a tip-up 90° pulse flips the magnetization back to the longitudinal plane (see Fig. 1). As the spin-lock amplitude B_1 approaches zero, the T1ρ relaxation time converges to the T2 (spin-spin) relaxation time.

The relaxation of the T1ρ signal can be described by the equation:

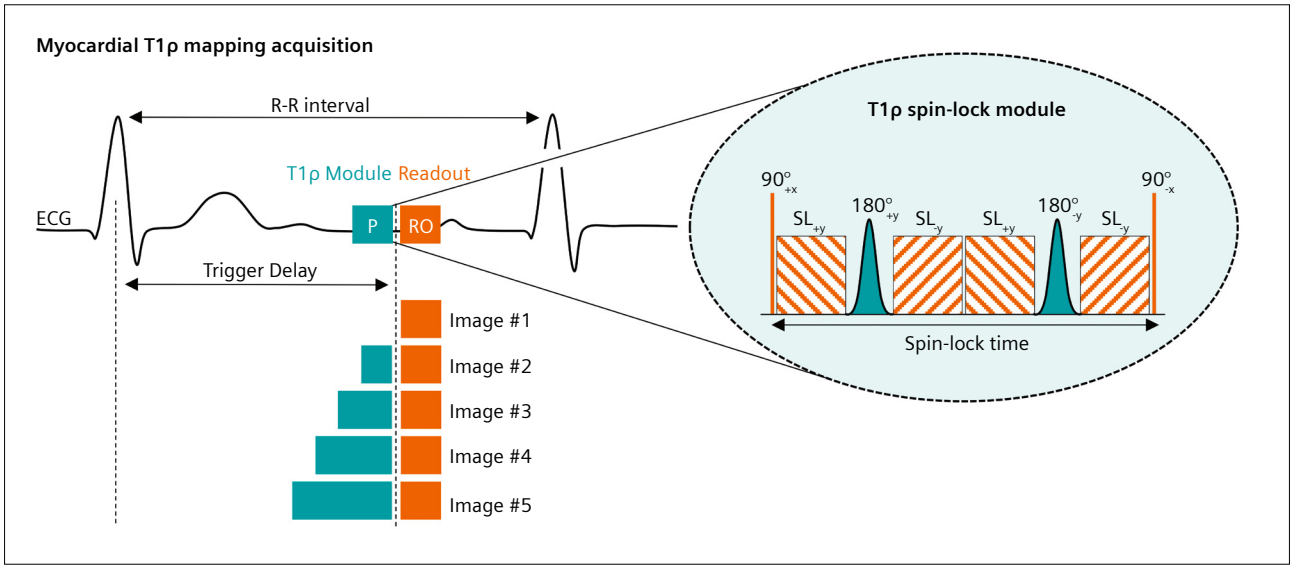
$$S(TSL) = M_0 \cdot e^{-\frac{TSL}{T1\rho}}$$

Here, S denotes the signal intensity, TSL is the spin-lock time (in ms), and M_0 denotes the equilibrium magnetization. At clinical field strengths, spin-lock amplitudes range from 100 to 500 Hz, and TSL varies from 0 to 100 ms, depending on the specific tissue under examination.

Tips and tricks for successful T1ρ imaging and quantitative analysis

At Bordeaux University Hospital, we have successfully applied myocardial T1ρ mapping in over 200 studies, encompassing both ischemic and non-ischemic cardiomyopathies across acute and chronic stages.

The technical details of this prototype myocardial T1ρ mapping sequence[1] are described in detail in [9, 10]. Figure 1 outlines the standard cardiac acquisition scheme that we use. It employs an electrocardiogram-triggered pulse sequence to capture multiple images at various spin-lock times across the T1ρ decay curve. To ensure synchronization with the quiescent cardiac phase, particularly mid-diastole, the trigger delay is adjusted in practice. A two-heartbeat gap between acquisitions allows for full magnetization recovery, with flexibility based on the patient's heart rate. In 2-dimensional imaging, three (or more) short-axis slices (basal, mid-cavity, apical) are each routinely acquired within a single breath-hold, producing five T1ρ-weighted images per slice for the quantitative T1ρ map. At 1.5T, a balanced steady-state free precession readout with a minimum of five source images is recommended. A widely used T1ρ pattern involves the 4-spin-lock scheme interspersed with two 180° adiabatic refocusing pulses [4].



1 Myocardial T1ρ mapping framework, T1ρ pulse employed, and curve fitting process.

[1] *Work in progress. The application is currently under development and is not for sale in the U.S. and in other countries. Its future availability cannot be ensured.*

Table 1 presents typical imaging parameters for myocardial T1ρ mapping at 1.5T. Despite breath holding, it is possible that respiratory drift of the heart (average displacement of 5.1 ± 2.7 mm in a recent study of 30 adult patients), variations in diaphragmatic position during serial breath-holds, as well as fluctuating R-R intervals occur [9]. Consequently, adopting motion correction strategies (such as the Heart-Freeze Inline Motion Correction from Siemens Healthineers [11]) is strongly recommended to enhance the robustness and clinical acceptance of myocardial T1ρ mapping (Fig. 2).

For inline data processing, the generation of quantitative T1ρ maps involves a two-parameter pixel-wise curve fitting process. The resulting output includes both source and motion-corrected T1ρ-weighted images, along with T1ρ maps, which are then used for subsequent quantitative analysis. Protocol parameters tested on a 1.5T MRI scanner (MAGNETOM Aera, Siemens Healthineers, Erlangen, Germany) are provided in Table 1.

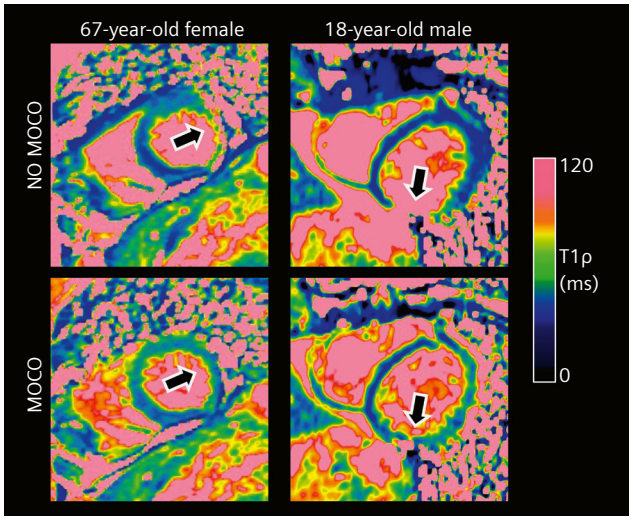
Sequence setting	Parameter range
Acquisition	Single-shot SSFP
Cardiac control	ECG triggering
Respiratory control	Breath-holding with non-rigid motion correction
Spatial resolution	1.4 × 1.4 mm^2 to 1.9 × 1.9 mm^2
Slice thickness	8–10 mm
Acquisition window	160–250 msec
Receiver bandwidth	900–1200 Hz/pixel
Flip angle	70°
Recovery heartbeats	3
Parallel imaging	GRAPPA 2 with 36 reference lines
Phase FOV	80%
Phase resolution	75%
Partial Fourier	6/8
Asymmetric echo	Weak
Dummy heartbeats	0
Distortion correction	Yes
k-space encoding	Linear
T1ρ module	$90_{+x} – SL_{+y} – 180_{+y} – SL_{-y} – SL_{+y} – 180_{-y} – SL_{-y} – 90_{-x}$
T1ρ number	5
T1ρ durations	0, 10, 20, 35, 50 msec
T1ρ spin-lock frequency	400–500 Hz
Scan time	13 heartbeats

Abbreviations: ECG, electrocardiogram; FOV, field of view; GRAPPA, generalized autocalibrating partially parallel acquisitions; SSFP, steady-state free precession

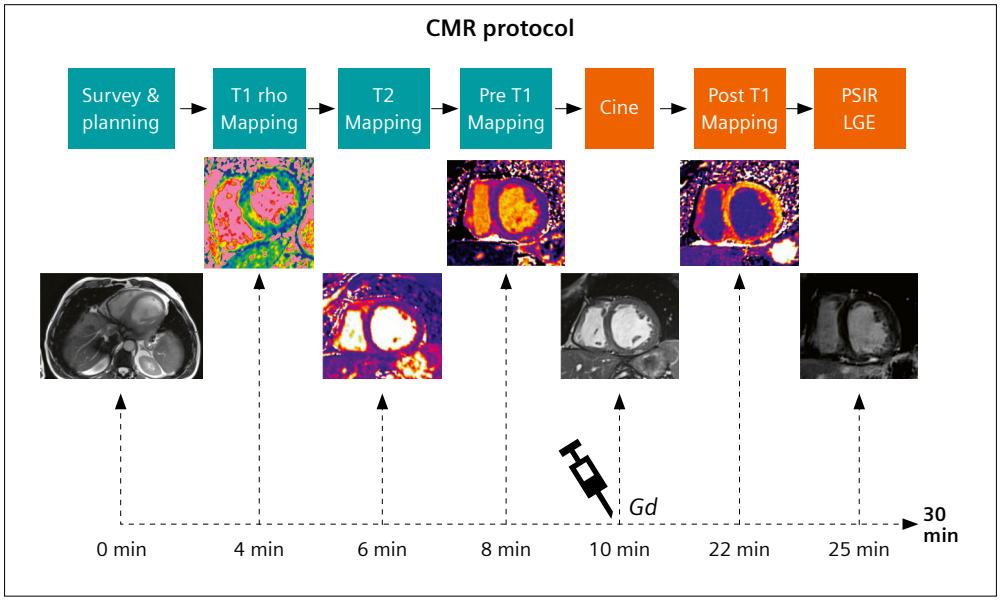
Table 1: Acquisition parameters for myocardial T1ρ mapping at 1.5T

The planning of the T1ρ mapping prototype sequence closely mirrors that of the T2 mapping sequence in the MyoMaps package, with the main difference being a slightly extended scan time due to the collection of two additional single-shot images. Myocardial T1ρ mapping is therefore seamlessly integrable into your existing pre-contrast mapping portfolios, with the added convenience of being able to copy scan parameters from the T2 mapping sequence, and vice versa (Fig. 3). Below are some key protocol recommendations that follow the clinical recommendations on parameter mapping from the Society for Cardiovascular Magnetic Resonance [12]:

1) T1ρ values should be measured in the absence of contrast agents.
2) While in-plane motion correction is advisable if available, it is not a substitute for breath-holding.
3) Ensure proper adjustment of the main magnetic field shim (focused on the heart) and the center frequency to minimize off-resonance effects.
4) For a regular heart rhythm, we recommend collecting the images in mid-diastole (Fig. 4).
5) Regularly review the image quality during acquisition, monitor the heart rate and breath holding, inspect the source T1ρ-weighted images, and check for wrapping artifacts.
6) T1ρ maps will be presented in color, with a color look-up table specifically for T1ρ mapping provided. We suggest using a site-specific value range to emphasize abnormal areas.
7) Use any preferred on-site analysis software for region-of-interest drawing, left ventricular segmentation, and American Heart Association bullseye model generation. For quantitative analysis of maps, adhere to clinical recommendations for CMR mapping [12]. Given the need for standardization, always include as much information as possible in scientific articles, such as on-site normal reference range, T1ρ mapping sequence details, patient gender, age, and heart rate.
8) Similar to other CMR parameter mapping techniques, T1ρ values may be affected by partial volume effects, the presence of thin muscle, and residual breathing motion. These factors, which are especially prominent at the apical slices, may contribute to false positive diagnoses. In such instances, we recommend manually recalculating T1ρ values by measuring regions of interest from the non-motion-corrected source images. An Excel file for facilitating this calculation can be accessed here: magnetomworld.siemens-healthineers.com/clinical-corner/protocols/cardiovascular-protocols/cardiac-mr-mapping



2 Impact of motion correction on myocardial T1ρ map quality in two patients who had difficulties holding their breath.



3 Clinical CMR protocol at 1.5T including the prototype pre-contrast T1ρ mapping sequence.

4 Physio-Signal card of the T1ρ mapping sequence on a 1.5T MAGNETOM Aera scanner. Data is collected in mid-diastole, repetition time is set to the minimum, and a trigger pulse of three (two recovery heartbeats between acquisitions) is used for full magnetization recovery.

MRI safety considerations

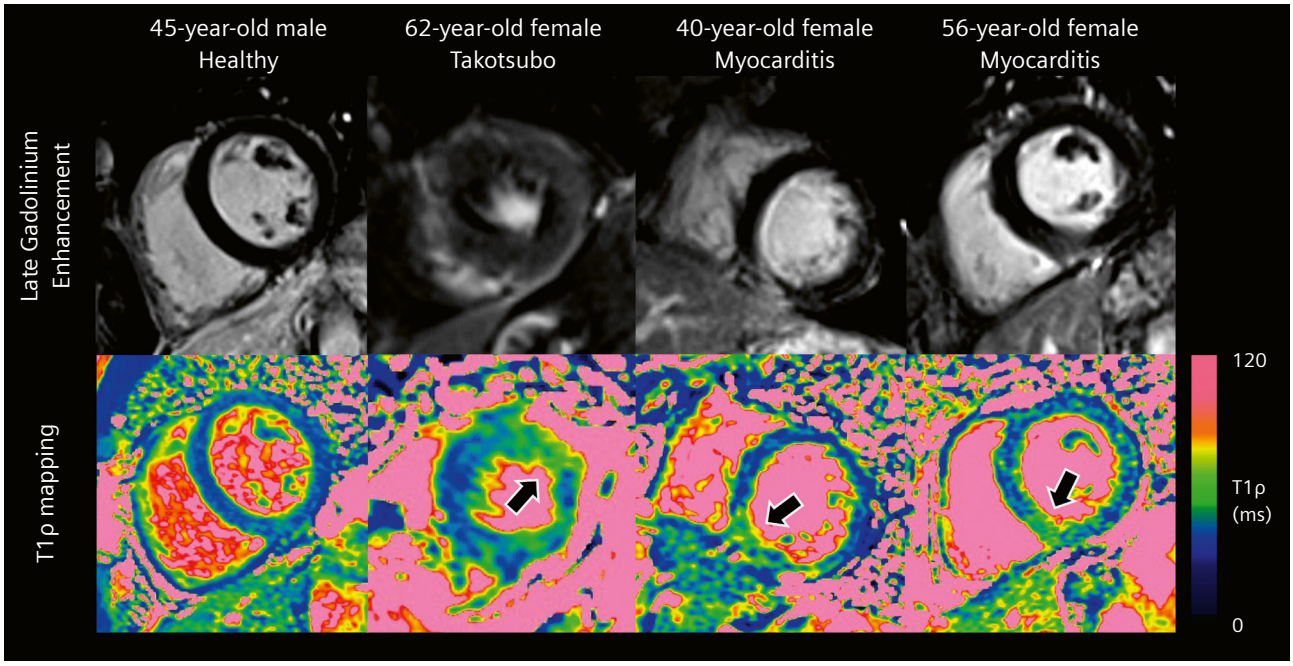
Since the presented T1ρ mapping sequence is used in the clinic, the specific absorption rate (SAR) has to be taken into account. In comparison to T2 mapping, T1ρ mapping is more SAR intensive due to the additional spin-lock modules. The SAR deposit depends on the duration and the frequency of the spin-lock pulse. In addition, the main magnetic field strength and the number of recovery heartbeats between acquisitions play a role in the calculation of the SAR. It should be further noted that employing a T1ρ preparation module with adiabatic refocusing pulses leads to a higher SAR value than a module with simple refocusing pulses. For this prototype T1ρ mapping sequence at 1.5T and the proposed parameters (see Table 1), the SAR values stayed in the range accepted for clinical applications. However, should the C2P be extended to 3D applications without ECG triggering, a re-evaluation of the SAR deposit will be necessary.

Clinical applications

As only very few studies have reported myocardial T1ρ values in patients [7, 10, 13–17], the release of this C2P will open the doors to a broader clinical exploration of this technology. All images shown in this article were acquired at Bordeaux University Hospital on a 1.5T MAGNETOM Aera scanner (with software version *syngo* MR E11C, Siemens Healthineers, Erlangen, Germany) using a dedicated 32-channel spine coil and an 18-channel body coil.

"Normal"/reference T1ρ values
Although various studies indicate normal T1ρ values around 50 ms at 1.5T, notable variability exists across centers, sequences, gender, and age.

Findings in Takotsubo and acute cardiomyopathies
In individuals experiencing acute myocardial injuries, our findings revealed a notable positive correlation between T1ρ and T2. In comparison to our control group, patients with acute injuries exhibited a substantial 36% increase in T1ρ. This observation highlights the fact that T1ρ could potentially serve as a valuable marker for the acute phase of myocardial infarction, similar to T2. The identification of T1ρ changes becomes accessible immediately after the acquisition of localizers, providing insights for the selection of sequences for the remaining protocol. Myocardial T1ρ maps and LGE images collected in three patients with acute myocardial injuries are presented in Figure 5.



5 Contrast agent-free myocardial T1ρ maps and post-contrast LGE images in a healthy subject, and in patients with Takotsubo cardiomyopathy and myocarditis. Elevated T1ρ values are observed in the three patients with cardiomyopathies (arrows).

Findings in hypertrophic cardiomyopathy

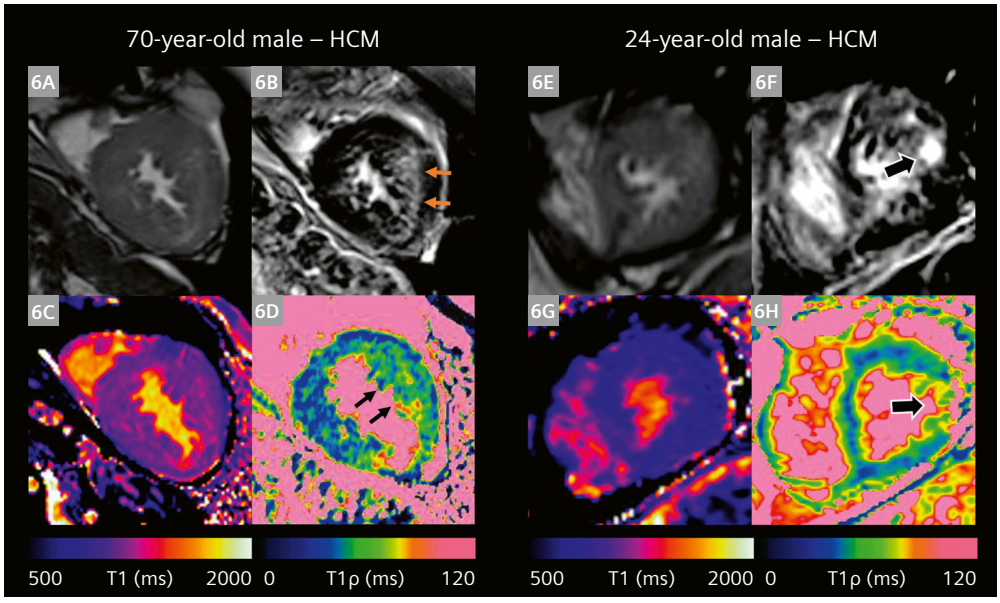
Case 1: A 70-year-old male patient with asymmetric hypertrophic cardiomyopathy and a maximal wall thickness of 18 mm. There is evidence of mid-wall fibrosis detected with LGE imaging, specifically in the mid anterior, inferolateral, and inferoseptal segments. An elevation of native T1 (1167 ms versus 1090 ms) and T1ρ (61 ms versus 49 ms) is also observed (Fig. 6A–6D).

Case 2: A 24-year-old male patient presenting with preserved left ventricular ejection fraction (56%) and asymmetric hypertrophic cardiomyopathy evidenced by a septal wall thickness of 18 mm. Focal LGE is observed on the apical inferolateral segment with slight deviations in T1 (986 ms vs. 1058 ms) and T1ρ values (46 ms vs 69 ms) (Fig. 6E–6H).

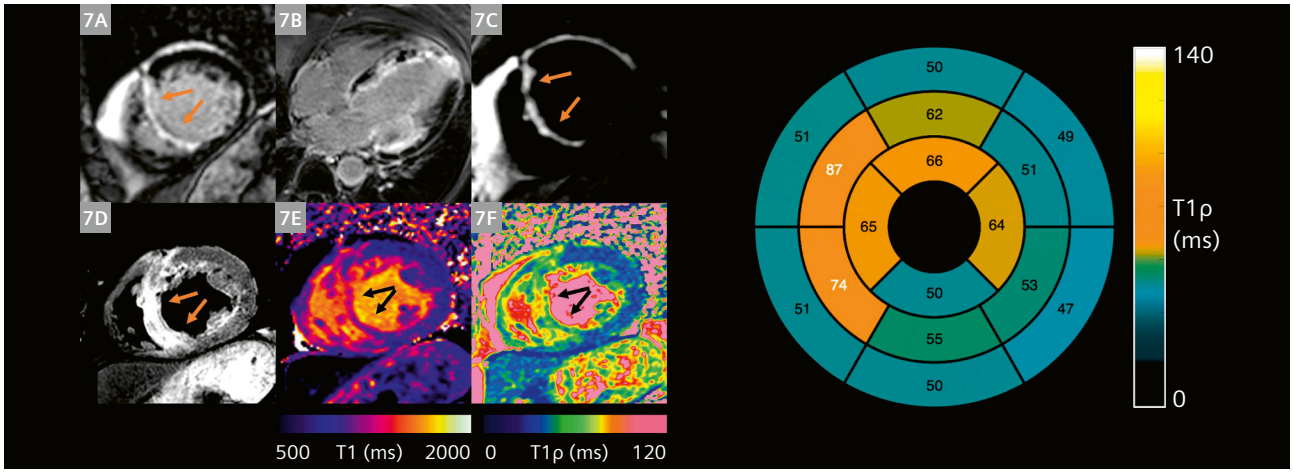
Two other studies have reported on the use of myocardial T1ρ mapping in hypertrophic cardiomyopathy [7, 13].

Findings in myocardial infarction

Case 3: A 70-year-old male patient presenting with a diagnosis of severe ischemic cardiomyopathy localized in the left anterior descending artery. CMR reveals extensive LGE evident in the basal, middle, and apical slices, characterized as transmural and non-viable. The patient had a significant impairment in the left ventricular function, with a notably reduced ejection fraction of 20%. Quantitative analysis revealed a measured native T1 of 1468 ms, compared to a T1 of 957 ms in a remote region. Additionally, T1ρ was prolonged to 106 ms in the affected region, as opposed to 50 ms in the remote area (Fig. 7).



6 Cases 1 and 2. Two male patients with hypertrophic cardiomyopathy imaged at Bordeaux University Hospital on a 1.5T MAGNETOM Aera system. **(6A)** and **(6E)** Short-axis cine images. **(6B)** and **(6F)** Phase-sensitive inversion recovery LGE images. **(6C)** and **(6G)** Native "MOLLI" T1 mapping. **(6D)** and **(6H)** Prototype T1ρ mapping[1].



7 Case 3. Myocardial T1ρ values in a 70-year-old male patient with myocardial infarction. **(7A)** Short-axis phase-sensitive inversion recovery (PSIR). **(7B)** Long-axis PSIR. **(7C)** Black-blood imaging [18]. **(7D)** Black-blood T2-weighted fast spin-echo. **(7E)** Native "MOLLI" T1 mapping. **(7F)** Prototype T1ρ mapping[1] and corresponding 16-segment model from the American Heart Association exhibiting elevated T1ρ values in the apical and medial segments.

Case 4: 60-year-old male patient presenting with reduced ejection fraction (46%) and notable findings on imaging consistent with myocardial infarction with non-obstructive coronary artery (MINOCA), indicating an ischemic event despite the absence of significant coronary artery obstruction. LGE reveals distinct fibrotic areas in the basal inferolateral segments. Quantitative analysis of native T1 values indicates an elevation in the affected area (1416 ms versus 1140 in remote). The T1ρ value is also prolonged to 84 ms (versus 53 ms in remote) in the affected segments (Fig. 8).

Several other studies have reported on the use of myocardial T1ρ mapping in myocardial infarction [6, 14].

What does the future look like?

As highlighted above, improvements in spatial resolution are imperative for advancing clinical detection and enabling better differentiation of lesions. Although myocardial T1ρ mapping is still in its early stages, 3D applications, especially in non-Cartesian formats, are emerging [19, 20]. The implementation of the "free-running" framework is pivotal for achieving cardiac- and respiratory-resolved (5D) T1ρ mapping [21].

Multiparametric mapping offers a comprehensive assessment of myocardial tissue characteristics. Magnetic resonance fingerprinting (MRF), including T1ρ mapping, will facilitate the simultaneous measurement of multiple parameters, enabling a thorough evaluation. An MRF framework for simultaneous T1, T2, and T1ρ cardiac mapping in a single 16-second scan has already been introduced by Velasco et al. [22].

Recent advances in the field of artificial intelligence also bring several opportunities such as quality control, motion correction, segmentation, and fully automated analysis, aimed at reducing manual workload and fostering clinical application [4, 23].

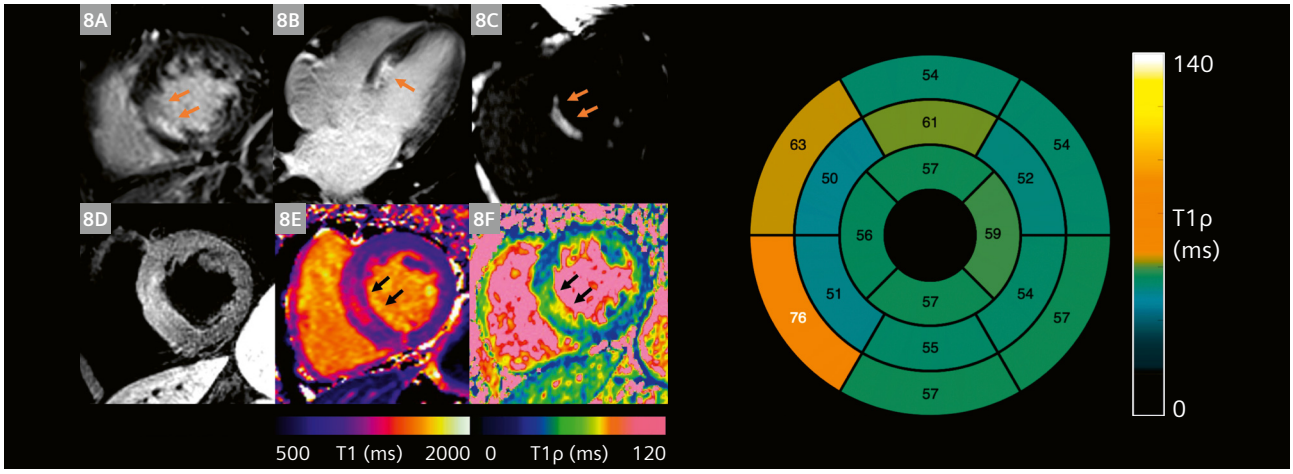
The widespread clinical adoption of T1ρ mapping faces challenges related to standardization and transferability. Collaborative efforts, such as using platforms like the C2P exchange from Siemens Healthineers, and sharing CMR protocols, are encouraged to enhance availability and standardization. Open access to data, reconstruction, and analysis code will further enhance reproducibility and accelerate the uptake of myocardial T1ρ mapping in routine clinical practice.

Conclusion

Magnetic resonance myocardial T1ρ mapping holds significant promise for the quantitative characterization of myocardial injuries. As we are just beginning to tap into the potential of this technology, the release of this C2P will hopefully facilitate gaining new and quantitative insights into myocardial disorders through collaborative, multicentric efforts.

Sequence availability

Our C2P sequence is currently available for sharing in *syngo* MR E11C on the C2P platform, so please feel free to take a look. Future releases in the NX version are planned and will be uploaded as soon as they are available. The .exar1 protocols for the 1.5T MAGNETOM Aera and the MAGNETOM Avanto (software version *syngo* MR E11C) are available to download at:
magnetomworld.siemens-healthineers.com/clinical-corner/protocols/cardiovascular-protocols/cardiac-mr-mapping



8 Case 4. Myocardial T1ρ values in a 67-year-old male patient with myocardial infarction with non-obstructive coronary arteries (MINOCA). **(8A)** Short-axis phase-sensitive inversion recovery (PSIR). **(8B)** Long-axis PSIR. **(8C)** Black-blood imaging [18]. **(8D)** Black-blood T2-weighted fast spin-echo. **(8E)** Native "MOLLI" T1 mapping. **(8F)** Prototype T1ρ mapping and corresponding 16-segment model from the American Heart Association exhibiting elevated T1ρ values in basal septal segments.

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