Reference Normal Values for Myocardial T1 and T2 Maps with the MAGNETOM Vida 3T System and Case Examples from Clinical Practice

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Abstract

T1 and T2 mapping techniques have become an important component of the study of myocardial tissue characterization in cardiac MR examinations. Easy to acquire, highly reliable, and quantifiable, thanks to their pixel-based conception, T1 and T2 maps provide radiologists and cardiologists with accurate information on the presence (or absence) and degree of myocardial tissue fibrosis, edema, or infiltrative processes. Their usefulness for the diagnosis and prognosis of patients with a variety of myocardial diseases has been extensively proven. Implicit in the nature of these techniques, however, is that the range of normal values is dependent on the field strength of the system and the software used for the acquisition. For this reason, it is recommended that every local MR facility investigates the range of reference values prior to their clinical application.

This paper presents preliminary data on myocardial T1 and T2 values from mapping performed on a series of healthy individuals at our institution using a MAGNETOM Vida 3T system. Also, as an illustration of the practical usefulness of this information, two clinical cases are presented in detail, with a particular focus on the relevance of T1 and T2 values for the diagnosis and management of patients.

Basis and rationale of T1 and T2 mapping techniques in cardiac diagnosis

By introducing techniques capable of providing information on the intrinsic magnetic properties of tissues, Cardiovascular Magnetic Resonance (CMR) has increased its potential to act as a comprehensive diagnostic tool. Over the last decade, T1 and T2 mapping sequences have made reliable pixel-based measurements of myocardial longitudinal and transverse relaxation times possible [1, 2]. Based, respectively, on a single breath-hold acquisition of a series of inversion recovery images with varying inversion time (TI), or a series of multiecho datasets with varying echo time (TE), T1 and T2 maps have become essential components of the body of information on tissue characterization that is available from a CMR study.

Extensive histologic validation has proven that there is a correlation between T1 mapping values and the presence and degree of diffuse interstitial myocardial fibrosis [3]. Due to deposition of proteins or other substances with magnetic properties (lipids or iron, for instance), T1 values are also sensitive to other pathological processes, either intra- or extracellular, such as myocardial

	T1 native (ms) n = 24	T1 post contrast (ms) n = 12	ECV fraction (%) n = 12	T2 (ms) n = 24
Mean ± SD	1230 ± 38.5	508 ± 50	25 ± 2.5	39 ± 2.2
Maximal normal value	1307	608	30	43
Minimal normal value	1153	408	20	34

Table 1: Results.

edema or infiltration [4]. One method that helps us to distinguish between them is a T1-derived calculation, the extracellular volume (ECV) fraction, which can be obtained from native and postcontrast T1 myocardial and blood pool maps, and also requires the hematocrit value [4]. T2 myocardial values, on the other hand, have proven to be extremely accurate in identifying tissue edema [5], such as that which is present in cases of acute coronary syndrome or in myocarditis.

The availability of mapping techniques is thus a key resource in current CMR studies, particularly since large multicenter studies have demonstrated the prognostic value of T1 and ECV in patients with dilated cardiomyopathy, of either idiopathic or ischemic origin [6, 7].

Reference normal values on a MAGNETOM Vida 3T system

While several authoritative documents on reference normal values have been published [8–10], we must bear in mind that T1 and T2 myocardial values depend not only on the tissue composition but also on the MR field strength and software used [4]. In light of this, the aim of the present paper is to present the preliminary information on normal values derived from our initial experience of using a MAGNETOM Vida 3T system, given that, to the best of our knowledge, no data have been published on this issue to date.

Population studied

T1 and T2 mapping sequences were performed on a series of 24 individuals (18 men), with a mean age of 50.8 ± 14.3 (range: 19–86) who, based on a CMR study, were deemed to be free of myocardial disease or any type of disorder capable of inducing an impairment of the left ventricular (LV) muscle. Reasons for the referral for CMR had been: a technically inadequate echocardiographic study during a routine checkup (10 cases); suspected (and not confirmed) LV hypertrophy (8 cases); suspected (confirmed or not) aortic dilatation (3 cases); and MR angiography of the pulmonary veins before an ablation procedure for paroxysmal atrial fibrillation (3 cases).

CMR acquisition parameters and analysis

Studies were performed on a MAGNETOM Vida 3T system with the XA10A software version, equipped with a 60-channel body coil.

T1 mapping was obtained by a single shot TrueFISP inversion recovery sequence prepared with multiple inversions combined with sampling and recovery periods. An ECG-gated, motion-corrected, short axis slice was prescribed at the mid ventricular level with the following parameters: slice thickness: 8 mm; TE: 1.06 ms; minimum TI 100 ms with 80 ms increments according to a 5(3)3 scheme; matrix size: 256 (phase res.: 66%); FOV (rectangular): 360 mm (phase FOV: 85.2%); bandwidth: 1085 Hz/px; flip angle: 35°. In the patients requiring a contrast study, and for whom a recent hematocrit value was available (n = 12), the sequence was repeated 15 minutes after a 0.2 mmol/kg dose of contrast (gadoteric acid) had been given.

T2 mapping was based on a GRE single shot FLASH readout with multiple T2 preparations and recovery periods. An ECG-gated, motion-corrected, short axis slice was prescribed at the mid ventricular level with the following parameters: slice thickness: 8 mm; TE: 1.28 ms; No. of T2 preps: 3 (0, 35, 55 ms); matrix size: 192 (phase res.: 75%); FOV (rectangular): 360 mm (phase FOV: 80.2%); bandwidth: 1184 Hz/px; flip angle: 12°. This sequence was obtained only before contrast was given.

T1 and T2 values were read using Siemens Healthineers *syngo*.via software on regions of interest (ROIs) with an area of 0.5–1 cm^2 manually placed at the level of the mid ventricular septum on the parametric map images obtained from the sequences detailed above. In the 12 patients with a contrast study and a hematocrit value available, ROIs were also drawn on the LV blood pool of the T1 map images and both tissue and blood pool measurements were repeated on the postcontrast images. In these cases, the ECV fraction was calculated using the equation [4]:

$$ECV = \frac{1/T1\ myoc\ post\ Gad - 1/T1\ myoc\ native}{1/T1\ blood\ post\ Gad - 1/T1\ blood\ native} \bullet 100\text{-hematocrit}$$

Results

The results of these calculations are presented in Table 1. Upper and lower normal values are determined as the mean value plus and minus 2 SD, respectively, as recommended [4].

Case examples

The availability of a range of normal values obtained locally provides a robust basis for these techniques to be applied in clinical practice. Two case examples from our experience are described below.

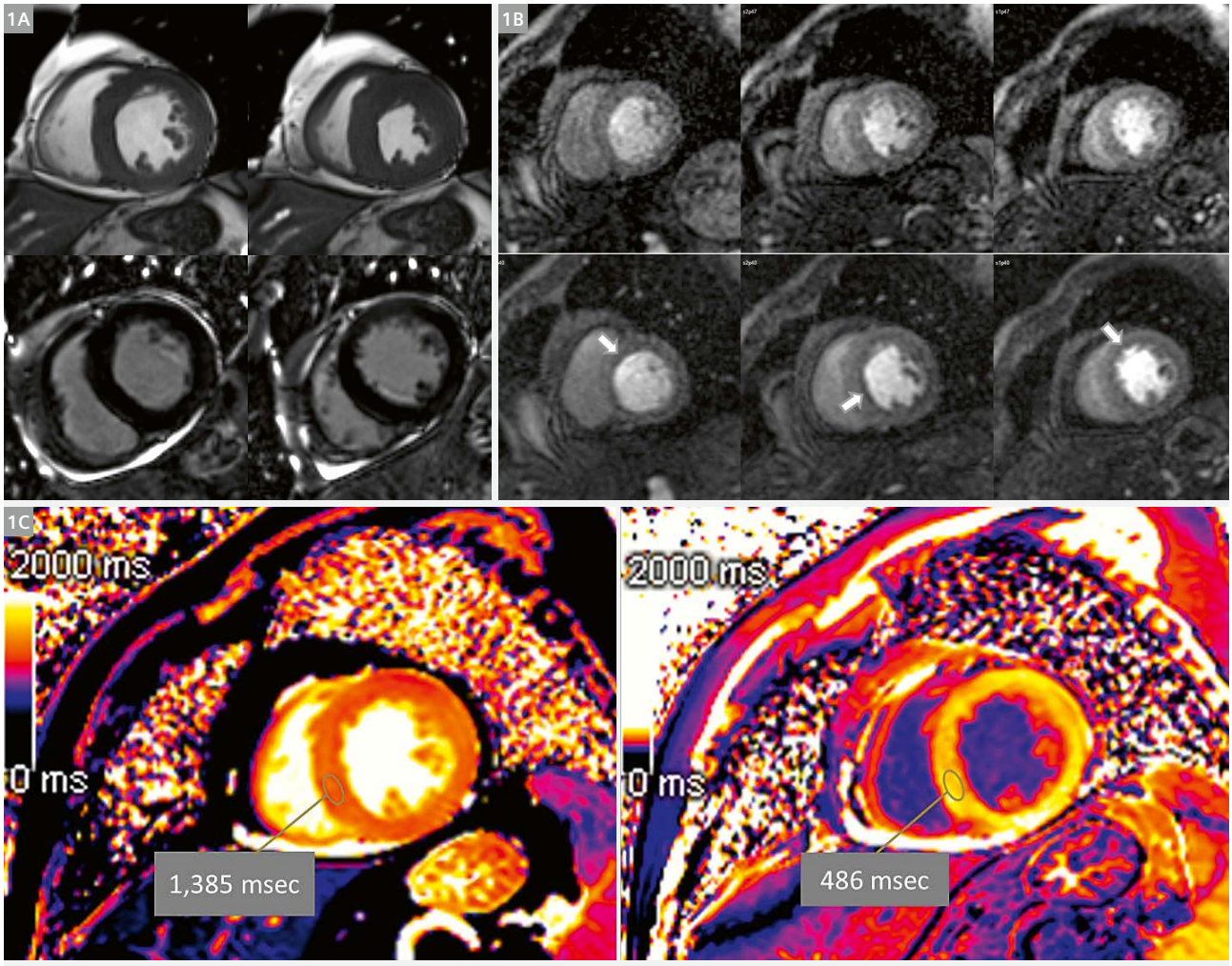

Figure 1:
(1A) End-diastolic (top left) and end-systolic (top right) frames from cine sequences obtained on a short axis mid ventricular plane, and delayed enhancement images (bottom row) obtained from the same patient 10 minutes after contrast injection.

(1B) Contrast first-pass perfusion studies performed at rest (top row) and at 4 minutes of an adenosine infusion (adenosine triphosphate at 160 mcg/kg/min) (bottom row). Arrows point to a diffuse, annular, subendocardial stress induced perfusion defect.

(1C) Native (left) and postcontrast (right) parametric T1 map images with ROIs showing the corresponding calculated myocardial T1 values.

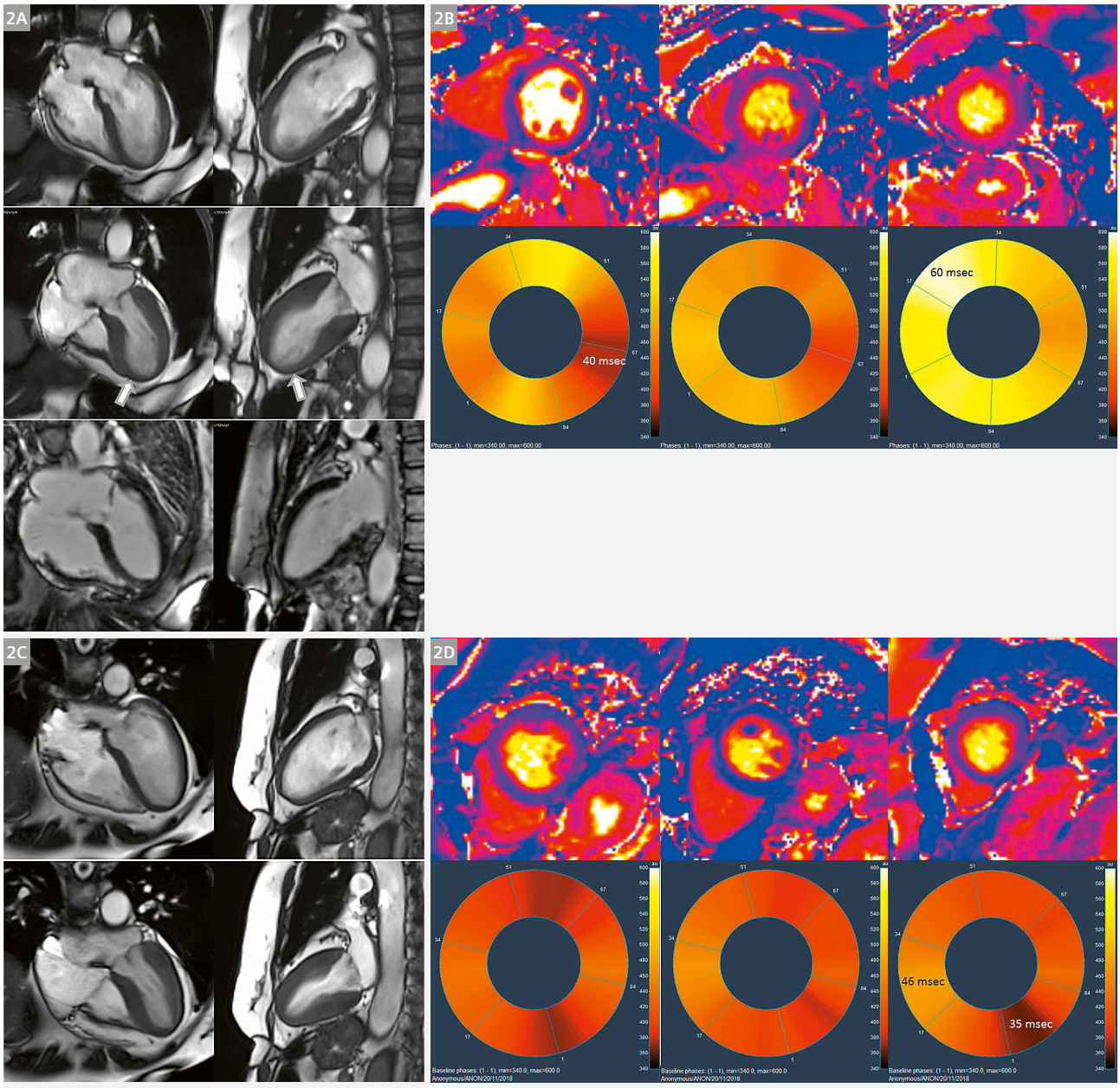

Figure 2:
(2A) End-diastolic (top row) and end-systolic (middle row) frames from cine sequences obtained in 4-chamber (left panels) and 2-chamber views (right panels): A marked dyskinesia of the LV apical region is seen (arrows). No apparent myocardial enhancement is observed on the same planes in IR sequences obtained 10 minutes after contrast injection (bottom row).

(2B) Parametric images from T2 map sequences obtained at basal, middle, and apical short axis LV planes (top row), and maps of the corresponding T2 values (bottom row, analyzed using Medis 7.1 QMass software[1]). Values within normal limits (< 43 msec) are observed only at the basal lateral region, while the septal anterior apical wall shows the highest readings.

(2C) Follow-up CMR study performed on the same patient. End-diastolic (top row) and end-systolic (bottom row) frames from cine sequences obtained in 4-chamber (left panels) and 2-chamber views (right panels) show recovery of the apical dyskinesia observed in the previous study (Fig. 2A).

(2D) Follow-up parametric images from T2 map sequences obtained at basal, middle, and apical short axis LV planes (top row), and maps of the corresponding T2 values (bottom row), both with the same orientation and analysis method as in the study shown in Figure 2B. Nearly complete normalization of values is determined compared with the previous examination.

[1] *The information shown refers to products of third-party manufacturers and are thus their regulatory responsibility. Please contact the third-party manufacturer for further information.*

The first patient, illustrated in Figure 1 (A–C), was a 74-year-old man with long-standing hypertension who presented with chest pain and signs and symptoms of heart failure. An earlier echo scan had shown LV dysfunction and angiography had shown non-obstructive coronary artery disease (CAD). CMR showed definite concentric LV hypertrophy with moderately reduced ejection fraction (estimated at 36%) and absence of any identifiable focal intramyocardial fibrosis in the delayed contrast enhancement study (Fig. 1A). A stress-induced myocardial perfusion defect was seen (Fig. 1B), with features consistent with diffuse coronary microvascular dysfunction. Finally, T1 mapping sequences (Fig. 1C) showed a relatively high native value (1385 ms) and, after a postcontrast study, also an abnormally high calculated ECV fraction (32%). The CMR study in this patient constitutes a full picture of an advanced case of hypertensive heart disease, with LV remodeling and dysfunction, microvascular CAD, and significant diffuse intramyocardial fibrosis and interstitial expansion. Interestingly, this patient did not present with focal fibrosis at the standard delayed enhancement study, a feature which could have erroneously led his cardiologists to make a less adverse prognosis.

The second patient (Figs. 2A–D) was a 72-year-old woman who was admitted with clinical and ECG data suggesting an acute coronary syndrome. After an angiographic study showed apparently normal coronary arteries and a region of gross apical dyskinesia, a CMR exam was requested to confirm the diagnosis of Takotsubo syndrome. The study of function and delayed enhancement (Fig. 2A) showed findings typical of apical dyskinesia in the absence of myocardial necrosis, while a T2 map study of the LV myocardium with extensive coverage (Fig. 2B) showed a global increase in values, particularly at the anterior apical region, indicating diffuse, but predominantly anterior, apical distribution of myocardial edema. A follow-up CMR study performed four weeks after the acute episode showed nearly complete resolution of the wall motion abnormality (Fig. 2C) and also of the myocardial edema (Fig. 2D), as we would expect to occur in Takotsubo cardiomyopathy patients with a favorable outcome.

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