Differences in Null Points between the Left and Right Ventricles in Contrast-enhanced Inversion-recovery MRI in Patients with Cardiac Diseases

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INTRODUCTION
Delayed contrast-enhanced MRI has been applied to myocardial infarction as well as myocarditis, sarcoidosis, and hypertrophic cardiomyopathy. In these clinical entities, late myocardial enhancement may reflect myocardial scarring, inflammatory lesions, and necrosis of the LV myocardium. Some preliminary studies have indicated the usefulness of this imaging for detection of right ventricular (RV) myocardial damage associated with arrhythmogenic right ventricular cardiomyopathy (ARVC). However, the null points of the RV myocardium have not been assessed on contrast-enhanced MRI. The purpose of this study was to compare the null points of the RV myocardium with those of the LV myocardium using inversion-recovery (IR) fast T1 mapping imaging (i.e., the Look-Locker sequence) in patients with a wide spectrum of cardiac diseases. In addition, the signal differences in the RV and LV myocardia were evaluated in delayed contrast-enhanced IR gradient-echo MRI.

METHODS
Twenty-six consecutive patients with cardiac diseases were recruited into this study. Twelve patients had hypertrophic cardiomyopathy, seven had idiopathic RV arrhythmia, five had myocardial infarction, one had a suspicious LV mass, and one had a cardiomyopathy of unknown origin.

MRI was performed using a 1.5 T unit with high performance gradients (Intera NovaDual, Philips). A five-element phased-array cardiac coil was used for signal reception, and vector electrocardiography was used as a retrospective cardiac-gating. Approximately ten minutes after the 0.15 mmol/kg gadolinium injection, the Look-Locker sequence was acquired using cardiac-gated IR multi-shot gradient-echo EPI with the following parameters: TR, 7.9 ms; TE, 3.9 ms; flip angle, 10 degrees; EPI factor, 7; in-plane resolution, and 3.2 x 2.8 mm. The IR pulse was achieved 3.9 ms after the R wave. This was a short-axis single section (mid-ventricular level), and the imaging data of 45 cardiac phases were acquired with this imaging. After determining the null point of the LV myocardium using the Look-Locker imaging, delayed contrast-enhanced IR gradient-echo MR imaging was performed using cardiac gating and breath-holding methods. The imaging parameters for this sequence were: TR, 9.3 ms; TE, 4.6 ms; flip angle, 10 degrees; number of shots, 20; and in-plane resolution, 1.6 x 1.8 mm. Sensitivity encoding and linear k-space filling were employed.

Regions-of-interest (ROIs) were placed on the non-enhanced inferior wall of the RV and LV, and the background outside the body. The ROIs included 2-4 pixels in the myocardium. The SNRs of the RV and LV myocardia were calculated, and the null point of the myocardium was defined as the lowest value of the SNR in the Look-Locker imaging. The interval between the null points of the RV and LV myocardia was noted. The differences between the null points of the RV and LV myocardia were evaluated in delayed contrast-enhanced IR gradient-echo MRI.

RESULTS
Using Look-Locker imaging, the null points of the LV myocardium ranged from 250 to 320 ms. The null points of the myocardium were equal between the RV and LV in 17 of the 26 patients, whereas in the remaining nine patients the null points of the RV myocardium were shorter than those of the LV myocardium by 17–132 ms (i.e., 1-3 phases). These nine patients consisted of four with hypertrophic cardiomyopathy, four with idiopathic RV arrhythmia, and one with a suspicious LV mass.

In delayed contrast-enhanced MRI, the SNR differences between the RV and LV myocardia were significantly larger in the nine patients who had intervals between the null points of the RV and LV myocardia (565 +/- 533 %; 86.6-1725) than in the 17 patients who had no intervals (159 +/- 85 %; 3.90-701.9, P = 0.0084).

DISCUSSION
This study demonstrated that the null points of the RV myocardium were shorter than those of the LV myocardium in nine of the 26 patients with cardiac diseases examined. In these patients, there were larger differences in the SNR of the myocardium between the RV and LV in the delayed contrast-enhanced MR images. These results indicate a possible difficulty in detecting late enhancement of the RV myocardium without adjusting the inversion time of the delayed contrast-enhanced MRI, especially in patients who have differences in the null points between the RV and LV myocardia. The shorter null points of the RV myocardium may be contributed to by histological features, including the more sparse myocardial tissues and lesser coronary blood flow. The cardiomyopathies might involve the RV myocardium resulting in a shorter null point. The non-histological factors could be the closer position of the RV to the receiver coils and a partial volume effect between the RV myocardium and chamber, although a signal correction algorithm was employed.

In conclusion, the null points of the RV myocardium were shorter than those of the LV myocardium in some patients with cardiac diseases. Look-Locker imaging should be performed to evaluate the null points of the RV myocardium accurately.

REFERENCES