

VIABILITY

The apparent inversion time for optimal delayed enhancement magnetic resonance imaging differs between the right and left ventricles

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Background. Delayed post-contrast magnetic resonance (MR) imaging involves suppression of signal from myocardium using inversion times (TI) between 150–225 ms, when the myocardium appears dark and fibrotic scar appears bright. We noticed that at a TI optimized for signal suppression of the left ventricle (LV), the right ventricle (RV) appeared brighter. **Purpose.** The purpose of this study was to evaluate the TI for signal suppression in RV compared to LV, and to try and identify the cause of this observation. **Methods.** We studied 31 patients (ages ranged from 17–79 years, 11 females) who had an MR scan on a 1.5 T GE scanner. Delayed post-contrast short-axis images were obtained 20 minutes after injection of 0.2 mmol/kg of intravenous gadolinium chelate. TI optimization was performed by acquiring a range of TI times within a single breath hold, in increments of 25 msec. The TI time that resulted in lowest signal for the RV and LV was recorded. **Results.** With the imaging sequence employed, the TI leading to LV signal suppression ranged from 150–225 ms. At the TI that resulted in LV signal suppression, the corrected signal from the RV was significantly higher as compared to the LV (29 ± 13 au vs. 15 ± 8 au, $p < 0.001$). The findings were similar using only the body coil. The TI required to suppress the RV was usually ≤ 150 msec. The observation persisted before and after gadolinium infusion. **Conclusion.** The TI for myocardial signal suppression appears to be different between LV and RV. Potential mechanisms include partial volume averaging with fat or blood pool (related to increased trabeculation) in the RV. Alternatively, increased blood pool signal (within Thebesian veins or arterioluminal communications) in RV compared to LV leads to altered TI times due to similar partial volume effects.

Key Words: Delayed hyperenhancement; MRI; Inversion time; Right ventricle; Left ventricle

1. Introduction

Delayed magnetic resonance (MR) imaging after infusion of gadolinium has been demonstrated to be an excellent technique to accurately distinguish between a fibrous scar and viable myocardium. This technique involves imaging 10–30 minutes after contrast injection with a breath hold inversion recovery gradient echo sequence (1, 2). Delayed hyperenhancement (DHE) of the ventricular wall has been shown to correlate with myocardial fibrosis, necrosis, or inflammation and is thought to be due to differential washout kinetics of the gadolinium chelate between the affected tissue and the intact myocardium (3). In patients with a chronic MI,

DHE can accurately locate and determine the extent and transmural of the infarct. Thus, because of high spatial resolution, high reproducibility, and predictive value (2), MRI may become a reference standard for assessment of myocardial viability.

In order to maximize the difference in MR signal between normal myocardium and scar tissue, it is necessary to suppress the signal from the normal myocardium. This is achieved using an inversion recovery pulse, and the optimal inversion time (TI) may vary between patients. We have observed that at a TI optimized for maximal signal suppression of the normal left ventricle (LV), the signal from the right ventricle (RV) appeared distinctly brighter and vice versa, a finding that has not been described before (Fig. 1). If this indeed were the case, it might be necessary to adjust the TI differently for the LV and the RV, depending upon the cardiac chamber of clinical interest. The purpose of this study was to evaluate this observation and attempt to determine if different TI times of the LV and RV may have an anatomical basis or may be due to technical reasons of the pulse sequence.

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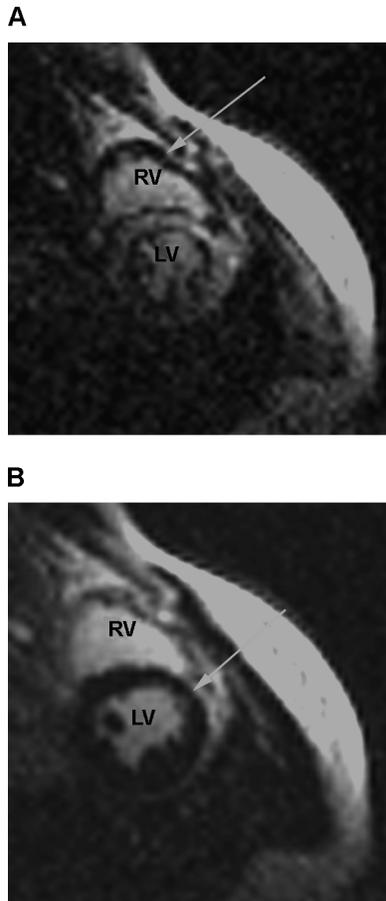


Figure 1. A. Short-axis delayed post-contrast image of the heart at TI = 150 ms demonstrating that the signal from right ventricle (RV) is significantly more suppressed than the signal from left ventricle (LV). B. Short-axis delayed post-contrast image of the same patient as in Fig. 1A at TI = 225 msec demonstrating that the signal from LV is significantly more suppressed than the signal from RV.

2. Methods

The study population consisted of 31 patients (ages ranged from 17–79 years, 11 females) referred to our cardiac MR laboratory for assessment of myocardial structure, function, and viability because of various suspected clinical conditions (dilated cardiomyopathies, arrhythmogenic right ventricular dysplasia, hypertrophic cardiomyopathy, ischemic heart disease, syncope). For the purpose of this study, we included consecutive patients who had no evidence of any myocardial abnormality on the delayed post-contrast images.

2.1. MR imaging technique

After obtaining informed consent, patients were imaged using a 1.5 Tesla MR scanner (General Electric Signa CV/i, Waukesha, WI). A 20-gauge intravenous catheter was placed in the antecubital vein for injection of gadolinium and

electrocardiographic leads were positioned. The cardiac phased-array coil was wrapped around the patient's chest and correctly positioned over the precordium. All cardiac images were obtained during a 12–15 heartbeat breath hold at end-expiration, averaging 10–15 seconds with adequate rest periods between breath holds (about 10–15 seconds). Each MR imaging session lasted 45 to 60 minutes in duration. The imaging protocol included sagittal, axial, and oblique scout images to localize the heart. 0.2 mmol/kg Gadolinium chelate (gadodiade, Amersham Health, NJ) was administered intravenously. LV/RV function and volumes were evaluated using cine imaging. After a 20-minute delay, delayed post-contrast images were obtained to assess DHE.

2.2. Delayed post-contrast imaging protocol

Twenty minutes following the contrast bolus, TI optimization for LV and/or RV was performed with a low resolution inversion-recovery prepared gated fast gradient echo pulse sequence that acquired a range of TI times, in increments of 25 msec, starting at 100–125 msec. This was achieved in one to two breath holds. Pulse sequence parameters were as follows: TR 5.1 msec, TE 2 msec, flip angle 20°, field of view 40 cm, matrix size 256 × 128, bandwidth 62.5 kHz, inversion pulse 180°, and 8-mm slice thickness with 0 gap. This generated a pixel size of 4.875 mm and a voxel size of 39 mm³. The TI time that resulted in lowest signal for the RV and LV, estimated visually, was recorded in increments of 25 msec. In five additional patients (four males, ages 28–52 years, similar inclusion criteria as the main study population), we also acquired the images prior to the administration of the contrast agent.

In order to account for the brighter signal in the RV wall (due to its proximity to the receiver coil) as compared to the LV wall (septum), we used the following formula to obtain corrected RV signal (4):

$$\text{Signal Intensity (RV free wall)} \times \text{Signal Intensity (blood pool in the LV adjacent to the septal wall)} / \text{Signal Intensity (blood pool in the RV adjacent to the RV free wall)}$$

In five patients, we also obtained higher-resolution delayed-enhancement images using a similar inversion-recovery prepared gated fast gradient echo pulse sequence. Eight to ten short-axis cross-sections of the ventricle of interest, to ensure entire cardiac coverage, were acquired. Pulse sequence parameters were as follows: TR 5.1 msec, TE 2 msec, flip angle 20°, field of view 36 cm, matrix size 256 × 192, bandwidth 15.6 kHz, inversion pulse 180°, and 8-mm slice thickness with 0 gap. This generated a pixel size of 2.65 mm and a voxel size of 21 mm³.

3. Statistics

Values of signal intensities are reported as mean ± SD. Paired t-testing was used to compare between means of signal

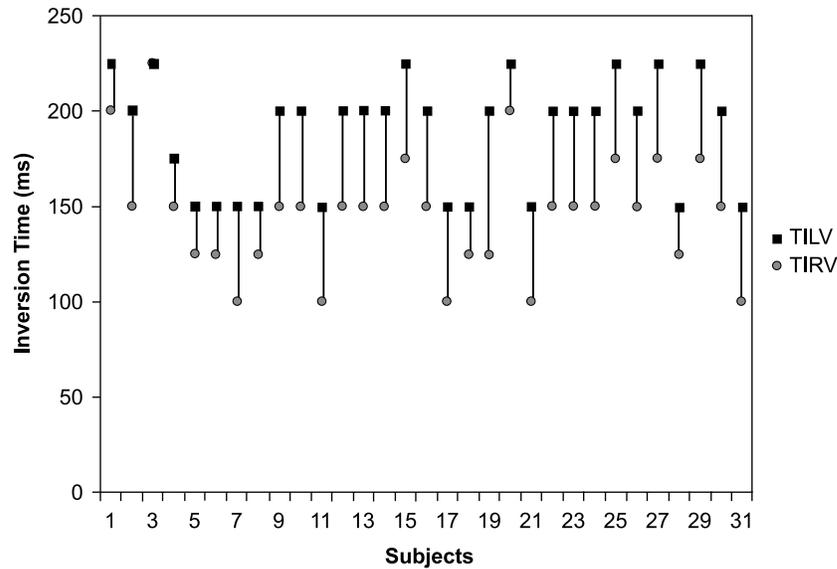


Figure 2. Scatter plot demonstrating the distribution of inversion times (TI) of the left and right ventricle (LV and RV) in all the subjects.

intensities between RV and LV. A p -value of < 0.05 was considered significant.

4. Results

All the patients completed the MR examination without complications. In the present study using the current sequence, the optimal post contrast TI leading to maximal LV signal suppression ranged from 150–225 ms in all the patients. At the TI that resulted in optimum LV signal suppression, the corrected signal from the RV free wall was significantly higher as compared to the LV (29 ± 13 au vs. 15 ± 8 au, $p < 0.001$). Subsequently, we obtained the same images using only the body coil and found that the difference in TI times for LV and RV persisted.

At the TI that maximally suppressed signal from the RV free wall, the LV showed suboptimal suppression. The TIs required to suppress the RV free wall signal were usually ≤ 150 msec. The TIs obtained from the LV and RV in all the patients are graphically displayed in Fig. 2. In order to determine if the TI time differences were due primarily to a difference in distribution of gadolinium between the RV and LV, five additional patients were also examined before and after the administration of gadolinium. A similar pattern of TI time differences for the RV and LV persisted before, as well as after gadolinium administration (Fig. 3).

Because RV free wall is usually significantly thinner than the LV walls and surrounded by pericardial fat, in a given pixel/voxel, there might be partial volume averaging due to a combination of the RV and fat (which generally has a shorter TI time). In 10 patients in whom higher resolution images were obtained (thus reducing the combination of RV myo-

cardium and pericardial fat in a given pixel/voxel), we found that the TI for the RV remained ≤ 150 msec.

We calculated TI times for myocardium considering partial volume inclusion of both myocardium and fat in the same voxel. Using a T1 for fat of 250 msec, the TI is approximately 160–170 msec ($TI = 0.69 \times T1$). Similarly, 20 minutes after gadolinium infusion, T1 of LV myocardium is approximately 300 msec, resulting in a TI of 200–225 msec. In a given voxel, partial volume effects for epicardial fat and myocardium were estimated to be present in the range of 0% to 99%. This resulted in TI times of myocardium with partial volume inclusion of fat ranging from 160–225 msec, following an exponential relationship.

5. Discussion

This study was initiated after an observation of apparent differences in the TI time of the RV and LV. In 31 patients with no evidence of DHE, this study confirms an observed difference in the TI between the LV and the RV myocardium. The TI for the RV myocardium was usually shorter than that of the LV myocardium. Hence, at an optimal TI to suppress the LV signal, the RV appears brighter and vice versa.

Using body coil imaging, we were able to demonstrate that this difference in signal persisted, i.e., it was not due to the proximity of the RV to the phased array surface receiver coil. Our simple correction for the RV signal intensity to normalize it to the same level as the LV signal intensity was based on the common assumption that the sensitivity of a coil falls off linearly with distance. Ideally, one would correct for this by using a surface-fitting or a filtering technique as described by Murakami, Hayes, and Weinberger (4), but since we

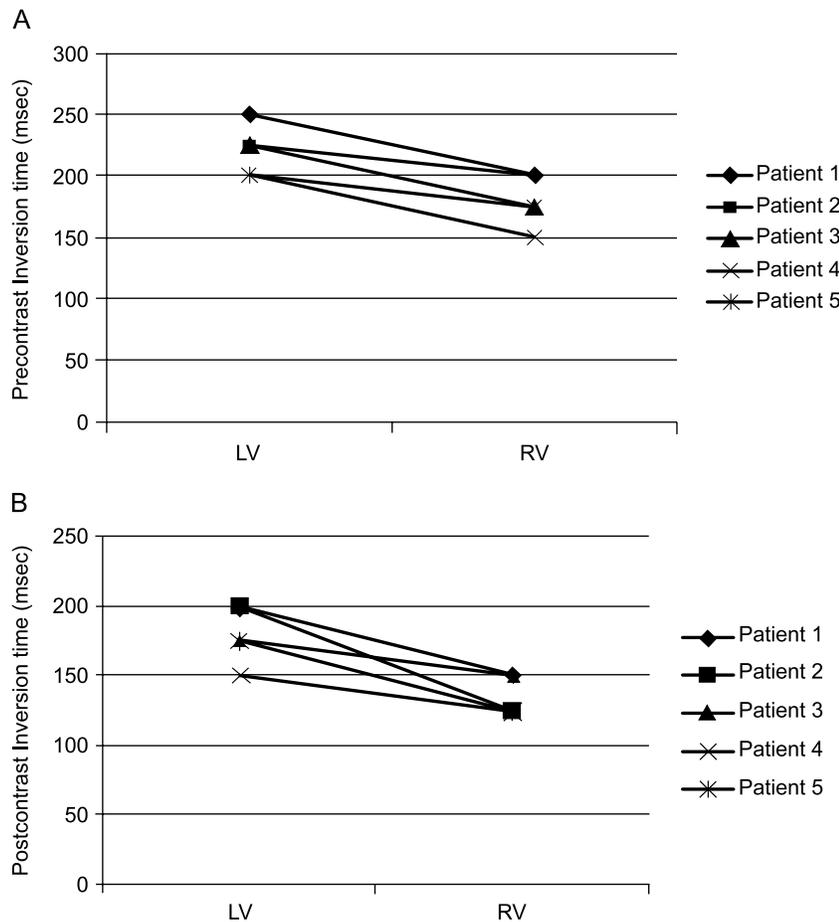


Figure 3. A) Scatter plot demonstrating the distribution of inversion times of the left and right ventricles (LV and RV) in five different subjects, prior to the infusion of gadolinium contrast. B) Scatter plot demonstrating the distribution of inversion times of the left and right ventricles (LV and RV) in the same five subjects, after the infusion of gadolinium contrast.

measured signal intensities in small regions of interest along an axis bisecting the RV and LV chambers, we simplified the task of fitting a surface through the image to fitting a straight line through the region of interests. This yielded our empirical correction equation.

We were also able to demonstrate that the changes in TI between LV and RV persisted before and after gadolinium administration. However, it is conceivable that the difference in the TIs between LV and RV may become more conspicuous with the administration of the contrast agent, but the increments of TIs (25 msec) utilized in the current pulse sequence were not sensitive to detect the increase in the TI in post-contrast vs. pre-contrast images. Thus, we feel that gadolinium might have a role in this observation, but it appears that there are other factors that need to be taken into account.

An additional important reason for this apparent difference could be partial volume averaging of fat and myocardium in the RV. The RV free wall ranges in thickness from 2–6 mm, and the pixel size for TI time optimization ranged from 2.65–4.875 mm. Partial volume effects with the inclusion of fat in the imaged voxel will

cause apparent TI shortening. The minimum expected TI time (post-contrast) for the RV is 160 msec (the TI time of a voxel with nearly all fat). Although the measurements of TI times were only in increments of 25 msec, the observed post contrast TI times of the RV ranged from 100–150 msec. One way to confirm or exclude the role of fat in the partial volume effect is to incorporate fat suppression into the imaging sequence.

Finally, it could be related to the differences in anatomical constitution of the RV and the LV myocardium or different accumulation of the gadolinium contrast agent. It has been described that there is a significantly higher number of Thebesian veins in the RV myocardium as compared to the LV myocardium (5). Also, arterioluminal communications (communication between the coronary arteries and ventricular chamber) were identified only in the RV myocardium and not in the LV myocardium. Hence, it is conceivable that there is an increased blood pool signal within the Thebesian veins and these arterioluminal communications traversing the RV myocardium as compared to the LV. This could lead to shortened TI in the RV as compared to the LV.

Delayed post-contrast MR imaging is being increasingly used for differentiation between a fibrous scar and viable myocardium. In fact, it is fast becoming a reference standard for evaluating the transmural extent of an infarct and assess for myocardial viability. Also, DHE might have a role in other disorders that also result in fibrosis or inflammation and could involve the RV (e.g., RV infarcts associated with inferior myocardial infarcts, hypertrophic cardiomyopathy (6), sarcoidosis, or myocarditis). Recently, it was also demonstrated that there is evidence of DHE in the RV in patients with arrhythmogenic RV dysplasia (ARVD) due to the presence of fibrofatty infiltration of the RV (7).

Typically, TI times are chosen for optimal suppression of the normal LV. In certain circumstances, it appears that adjustment of TI times to optimize RV pathology may be warranted. With the development of new inversion prepared cine sequences, we should be able to obtain a large number of slices at different TI times, albeit at different phases of the cardiac cycle. T1 mapping techniques have also been developed, but generally are low resolution images that are suboptimal for the very thin right ventricle. Hence, we feel that it is important to pay attention to the differences in TI between LV and RV and adjust it according to the clinical question and the relevant chamber.

6. Limitations

Based on the current study, we have not elucidated the nature of the difference in the TI between LV and RV. The best way to confirm this observation, we feel, is to measure the TI time of the LV and RV of a pathology specimen at different contrast doses, at different time points, and in different regions of both the LV and RV. Our observations suggest that this could be due either to intrinsic differences in the LV and RV, or that current MR methods result in significant partial volume averaging with blood pool or pericardial fat signal. Since the RV wall is extremely thin, partial volume effects in the z-axis direction (long axis of the heart) could not be eliminated while maintaining adequate signal-to-noise ratio. Also, we did not have pathologic confirmation of normal RV tissue. We

have only performed these studies on the MR scanner from one manufacturer, and it is possible that this observation is a consequence of the pulse sequence or the phase acquisition order.

7. Conclusion

The optimal TI for the suppression of LV myocardial signal is approximately 50 msec longer than that of the RV. Potential explanations may be partial volume effects due to close proximity of pericardial fat to the thin walled RV. It could also be due to increased blood pool signal within the RV wall due to a greater number of Thebesian veins and arterioluminal communications. In either case, it may be necessary to optimize TI separately for LV and RV, depending upon the clinical question and the cardiac chamber of concern.

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