Myocardial Wall Velocities Measured by Navigator-Echo Gated MR PVM in Patients with Dyssynchrony

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Introduction: Heart failure affects approximately 25 million people worldwide. Approximately 30% of heart failure patients develop an intraventricular conduction delay that causes the ventricles to beat dssynchronously. This dyssynchrony further contributes to decreased cardiac function and adverse ventricular remodeling. Treatment for dyssynchrony is highly invasive and involves subcutaneous implantation of a pacemaker and trans-venous placement of cardiac pacing leads. Approximately 30% of patients who receive pacemakers do not respond to the treatment [1]. Therefore, a better method is needed for accurately identifying dssynchrony patients. Current diagnostic methods for selecting patients for dyssynchrony treatment are surface electrocardiogram parameters or systolic contraction parameters based on Tissue Doppler Imaging (TDI). However, TDI can only accurately measure apex-to-base velocities within the myocardium. Measurement of myocardial tissue velocity by magnetic resonance (MR) phase velocity mapping could provide a complete three-dimensional description of myocardial motion and could potentially overcome the limitations of diagnostic methods based on TDI and ECG parameters.

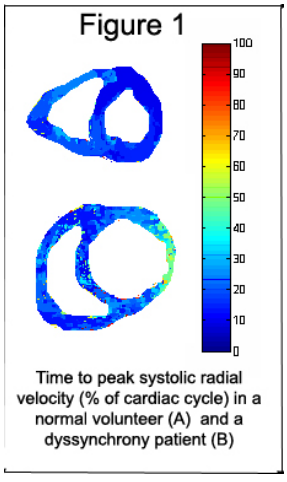
Purpose: The purpose of this study is to quantify three-dimensional (radial and longitudinal) myocardial velocities in normal volunteers and patients with dyssynchrony using navigator-echo gated MR phase velocity mapping. Systolic contraction velocities were compared between normals and patients.

Methods: Ten normal volunteers (ages 28.6+/-7.72) and ten patients (ages 61.8+/-15.63) with asynchrony (QRS >120 msec and LVEF < 35%) participated in the study. MRI scans were performed on a Philips Medical Systems Intera CV scanner. MR phase velocity maps were acquired in the short axis orientation with the slice positioned at 70% of the distance from the apex to base. A segmented, navigator-echo and ECG-gated sequence was used to acquire velocity in three orthogonal directions. In-plane resolution was 1.1 to 1.4 mm, slice thickness was 8 mm, and the VENC value of 20 cm/sec was used to encode velocities of the myocardial tissue. Presaturation slabs were used on each side of the slice to eliminate signal from fast flowing blood, which has a large phase shift. The first image was acquired 74msec after the R-wave trigger (due to the navigator pulse) and temporal resolution for the other frames was 35msec. Velocities were encoded in an interleaved fashion with reference scans and each velocity encoding direction acquired in successive heart beats. Navigator-echo gating and interleaving ensured that images from each velocity direction were correctly registered for post-processing.

MR phase velocity maps were processed off-line using an in-house developed MATLAB program. Background phase error was removed using a least-squares fitted plane. In-plane velocities were converted into radial velocities, with systolic motion toward the center of the LV blood pool defined as positive and diastolic relaxation defined as negative. For the through-plane velocity, motion toward the apex was defined as positive. Peak systolic velocities for 8 x 8 mm regions of interest in the septal, lateral, anterior and inferior walls were measured and averaged for each subject. Maps of time to peak systolic velocity were created for volunteers and patients. Time to peak systolic velocity (as a % of the cardiac cycle) was also computed for each ROI.

Results: MR phase velocity scans were successfully acquired in all patients and volunteers. Peak systolic velocities were significantly lower in patients than in normal volunteers in both the radial and longitudinal directions. Peak systolic radial velocity was 3.59 +/-1.65 cm/s for patients vs. 5.21+/-2.22 cm/s for normal volunteers ($p<0.001$). Peak systolic longitudinal velocity was 6.30 +/-2.7cm/s in patients vs. 7.89 +/-3.1 cm/s in normal volunteers ($p<0.05$).

The times to peak systolic radial and longitudinal velocities were significantly delayed in the lateral wall of dyssynchrony patients, Figure 1. Peak systolic radial velocity in the lateral wall occurred at 15.8 +/-0.1 % of the cardiac cycle in normal volunteers and 26.9+/-0.1% of the cardiac cycle in dsyssynchrony patient ($p<0.001$). Peak systolic longitudinal velocity in the lateral wall occurred at 11.1 +/-0.01 %of the cardiac cycle in normal volunteers and 24.9+/-0.08% of the cardiac cycle in dsyssynchrony patients ($p<0.001$). No significant differences in time to peak systolic velocities were observed between normal volunteers and dsyssynchrony patients in the septal, anterior, or inferior walls.



Conclusions: MR phase velocity mapping was successfully used to measure radial and longitudinal velocities in both normal volunteers and dyssynchrony patients. Systolic contraction velocities were higher in normal volunteers than in patients with dyssynchrony. Time to peak systolic velocity in the lateral wall was significantly delayed in patients compared to normal volunteers. Since MR phase velocity mapping can measure the complete 3-dimensional motion of the myocardium, it may help in evaluating patients with dyssynchrony.

References

[1] Auricchio, A., et al.. Am J Cardiol, 1999. **83**(5B): p. 130D-135D.