

A novel method for assessment and quantification of left ventricular dyssynchrony

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INTRODUCTION: Synchronous contraction and relaxation of the myocardium is required to optimize cardiac function [1]. Regional timing of contraction and relaxation is *dyssynchronous* in many patients with severe congestive heart failure [2,3]. Cardiac resynchronization therapy (CRT) is a relatively new treatment for dyssynchronous heart failure that results in both acute and long-term benefits [4,5]. Patients are currently selected for CRT using surface electrocardiogram QRS duration as a measure of dyssynchrony. However, up to 30% of patients selected for CRT based on ECG criteria show no improvement [5].

Directly imaging the myocardial wall with phase contrast MR (PCMR) can quantify mechanical dyssynchrony and potentially improve the CRT response rate. Many parameters to quantify dyssynchrony have been proposed, yet no single methodology has emerged as a widespread standard. Most investigators utilize “time-to-peak” analysis where the time from the electrocardiogram R-wave to the peak systolic velocity of one ventricular wall is compared to that of an opposing ventricular wall [6,7]. This analysis utilizes only a single point from all data collected throughout the cardiac cycle and assumes that the peak systolic velocity is the most clinically important criterion of ventricular synchrony.

We have developed a mathematical method to calculate a temporal delay between two myocardial tissue velocity profiles using a cross-correlation (XC) function to quantify dyssynchrony based on data collected *throughout* the cardiac cycle (not just peak values). We hypothesized that XC delay between two velocity curves would provide a more accurate measure of dyssynchrony and be better able to separate subjects with dyssynchrony from normal volunteers than methods based on “time-to-peak” analysis.

PURPOSE: To compare the XC delay and existing measures of dyssynchrony, including the septal-to-lateral delay (SLD) and the standard deviation of times-to-peak (T_{SD-6}), in a group of heart failure patients scheduled for CRT and a group of normal volunteers without cardiac disease.

METHODS: Myocardial velocity maps were acquired on a 1.5T Philips Medical Systems Intera CV MRI scanner using a cardiac coil. A segmented, navigator-echo and ECG-gated sequence was used to acquire three-directional velocities in a basal short axis slice of the left ventricle [8]. Presaturation slabs on each side of the slab were used to null in-flowing blood. VENC value was 30 cm/s, temporal resolution was 26 msec, and voxel size was 1.4 x 1.4 x 8mm. Velocity maps were acquired in 10 normal volunteers and 28 heart failure patients prior to CRT.

Velocities were converted into radial, circumferential, and longitudinal coordinate systems and averaged into six basal segments (anterior, anteroseptal, inferoseptal, inferior, inferolateral, and anterolateral) according to the AHA model. The septal wall was defined as the average of the anteroseptal and inferoseptal walls, and the lateral wall was defined as the average of the inferolateral and anterolateral segments.

Several measures of dyssynchrony were computed. Septal-to-lateral delay (SLD) was computed as the difference in time to peak systolic velocity between the septal and the lateral wall [10]. The standard deviation of times-to-peak in the six basal regions (T_{SD-6}) was computed as the standard deviation of times-to-peak systolic velocity in the six AHA basal regions [11]. The XC delay was computed between the septal and lateral curves by shifting one curve in time relative to the other curve and computing the correlation between the curves for each time shift. The time shift between the two curves that resulted in the maximum correlation value was defined as the XC delay between the septal and lateral walls.

RESULTS: The XC delay was the only parameter that consistently separated the normal and patient groups. P-values for the septal-to-lateral delay (SLD) between the normal and patient groups were 0.57 for the longitudinal direction and 0.11 for the longitudinal direction. P-values for the T_{SD-6} between the normal and patient groups were 0.54 in the radial direction and 0.19 in the longitudinal direction. The p-value for the XC delay between the normal and patient groups was <0.005 for both the radial and longitudinal directions.

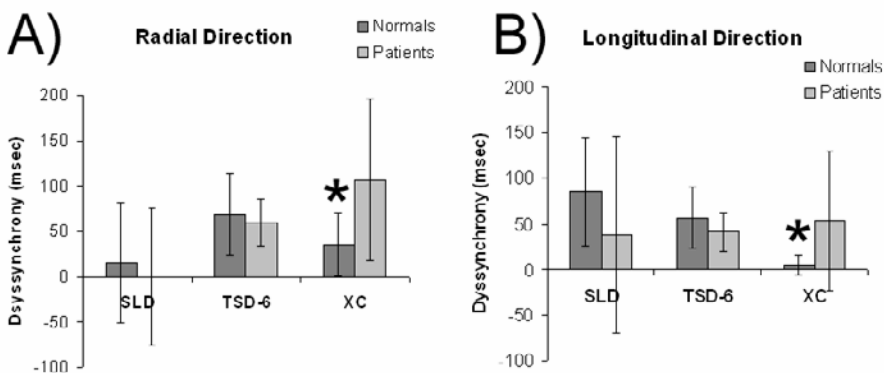


Figure 1: Dyssynchrony measured by the septal-to-lateral wall motion delay (SLWMD), the standard deviation of times-to-peak systolic velocity in six basal segments (T_{SD-6}), and the cross-correlation delay (XC delay) in the radial (A) and longitudinal directions (B). The XC delay was the only parameter that consistently separated the normal volunteers from the dyssynchrony patients. * denotes $p < 0.005$.

CONCLUSIONS: The XC delay provides a more robust measurement of dyssynchrony than parameters based on time-to-peak analysis. This may aid in the selection of patients for CRT.

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