Analysis of segmental diastolic asynchrony in patients with LV hypertrophy using tissue phase mapping

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Introduction: Diastolic dysfunction is an early indicator of cardiac disease and a common cause of heart failure. As regional left ventricular (LV) velocities are not distributed in a uniform manner in the healthy heart [1], a segmental analysis of myocardial motion is essential for the early diagnosis of altered LV motion. Tissue Phase Mapping (TPM) allows the quantitative segmental evaluation of myocardial velocities with high spatial and temporal resolution and full ventricular coverage [2]. The aim of our study was to analyze the detailed segmental distribution of parameters reflecting the magnitude and timing of LV contraction, shorting and rotation. TPM and analysis methods were applied in a study with 18 patients with LV hypertrophy due to hypertension and compared to findings in an age-matched group of healthy volunteers.

Methods: All measurement s were performed on a routine MR system (1.5T, Sonata, Siemens). Data acquisition consisted of a black blood prepared gradient echo TPM sequence (TR=6.9 ms; temporal resolution 13.8 ms; spatial resolution 1.3x2.6 mm; venc = ±15 cm/s in-plane, 25 cm/s through-plane) in 3 short axis slices (basal, midventricular, apical). Measurements included prospective ECG- and advanced navigator gating [3], view sharing and first-order flow compensation. 18 patients with LV hypertrophy due to hypertension (wall thickness >12 mm, age = 53.4±12.3), and 20 healthy age-matched volunteers (age = 51.3±3.9) were examined. Data post-processing (Matlab, The Mathworks, USA) included correction for translational motion and a transformation of the measured three-directional motion velocities into radial, rotational and long-axis velocities adapted to the LV anatomy. A 16 segment AHA model, including endo- and epicardial regions was used for segmental analysis. To quantify differences of LV function, systolic and diastolic peak and time-to-peak (TTP) velocities of radial and longitudinal velocities were derived from the velocity time course of each segment.

Results: Patients demonstrated a marked reduction and delay of mean diastolic peak velocities compared to healthy controls although there was no difference in the heart rate between the two groups (Table 1). Figure 1 and 2a represent the diastolic delay of segmental peak long- axis and radial velocities. Diastolic radial velocities of patients were delayed in nearly all segments (Figure 1), whereas long-axis velocities showed a more complex delay in basal, midventricular and septal regions compared to healthy volunteers (figure 2a). For both velocity components, segmental analysis showed not only a general delay of velocities but also an altered distribution of TTP, reflecting a change of diastolic performance within the LV. For radial velocities the healthy ventricle demonstrated a TTP gradient between basal and apical regions which was absent in inferior and septal segments of patients. For long-axis velocities the diastolic TTP clearly demonstrated an increased asynchrony within the LV in patients. In the healthy LV, diastolic peak long- axis velocities were highest in basal and lateral parts of the LV and lower in septal regions and toward the apex. These intraventricular differences were substantially reduced in the patients (see figure 2b).

Discussion: Patients with hypertensive LV hypertrophy demonstrated extensive alterations in magnitude, timing and distribution of segmental myocardial velocities during diastole. The importance of the exact temporal and spatial distribution of myocardial contraction and expansion within the LV, so called myocardial synchrony, is a matter of increasing attention in cardiology. Diastolic asynchrony begins to influence therapeutic decisions and even outcome and prognosis in heart diseases seem to be correlated to synchronous myocardial performance. To date, however, the complexity of segmental myocardial performance is poorly understood. New imaging methods such as TPM and the detailed analysis and illustration of all aspects of the segmental myocardial performance may help to improve the understanding of the complex effects of heart disease.

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