

Regional Frank-Starling Effect Contributes to Larger Peak Strain in Lateral Free Wall in the Healthy Human Heart

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Introduction: In the healthy human heart, peak strains are regionally different, the lateral wall reaching higher peak strains than the septum [1]. We hypothesized that the regional differences in peak strain are caused by regional differences in the amount of prestretch due to the atrial contraction: a regional manifestation of the Frank-Starling mechanism. The purpose of this work was to determine regionally the left ventricular (LV) prestretch induced by the atrial contraction, and to relate this prestretch to regional function.

Methods: Imaging. Eleven healthy volunteers (age 41±13 year, 7 male) were studied using a 1.5 T Siemens Sonata system, with a 6-elements phased array receiver coil. Steady state free precession imaging with myocardial tagging (LISA-SSFP) [2] was used to acquire 5 short-axis complementary tagged images (CSPAMM) with 14 ms temporal resolution.

Post-Processing. Circumferential strain (ε_c) curves were calculated using the Harmonic Phase method [3], for the infero-septal (IS), antero-septal (AS), anterior (AN), antero-lateral (AL), postero-lateral (PL) and inferior (IN) regions. We assume that the LV fibers have their true resting lengths during diastasis. Thus the remaining plateau strain during diastasis was used as derived measure for the amount of prestretch due to the atrial contraction. The beginning of the diastasis (period after passive filling and before onset atrial contraction) was defined as the first zero-crossing of the strain-rate following fast relaxation, and its end was defined at 200 ms before the next ECG-R wave (Fig 1). Regional systolic function was quantified by the peak circumferential shortening (peakCS). Data was averaged over all subjects, after which regression analysis was used to study the relation between the regional amount of prestretch and function.

Results: The long tag persistence with LISA-SSFP tagging allows strain analysis throughout approximately the entire cardiac cycle (Fig 1). The regional pattern of prestretch caused by the atrial contraction shows more prestretch in the lateral wall (Fig 2A, $P < 0.001$), and is similar to the pattern in peakCS (Fig 2B). A linear relation between peakCS and prestretch was found: $y = 1.3x + 16.3$, $R^2 = 0.58$ (Fig 3, $P < 0.001$).

Discussion: The lateral free wall may be stretched more than the septum by the atrial contraction because the lateral wall is thinner, and because the septum experiences counterbalancing effects from the right and left ventricular blood pools.

Conclusion: LV-prestretch is not uniform in the human heart and causes (by the Frank-Starling effect) larger strain values in the lateral wall.

Literature:

- [1] Moore CC et al. Radiol. 2000; 214:453-466.
- [2] Zwanenburg JJM et al. Magn Reson Med. 2003; 49:722-730.
- [3] Osman NF et al. Magn Res Med 1999; 42:1048-1060.

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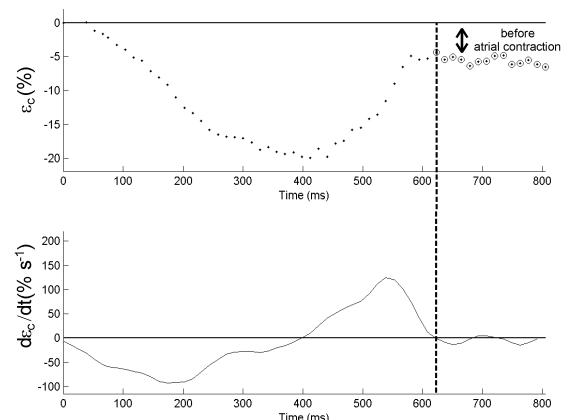


Fig 1. Example of circumferential strain (ε_c) (top) and strain-rate (bottom) obtained with SSFP myocardial tagging. The average strain plateau at diastase (encircled data points) is used as measure for the amount of prestretch induced by the atrial contribution. (RR duration of this subject approx. 1100 ms.)

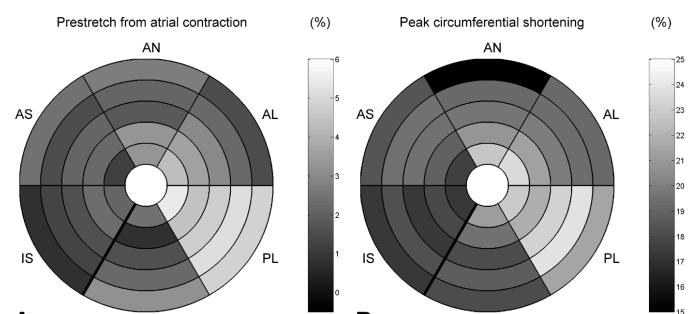


Fig 2. Bull's-eye maps for the healthy left ventricle, averaged over 11 subjects. A: prestretch due to atrial contraction. B: peak circumferential shortening.

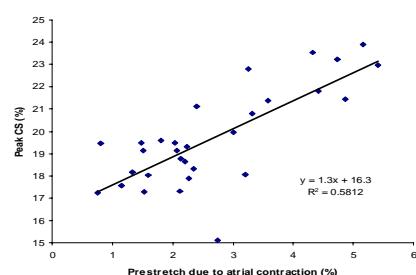


Fig. 3 Relation between peakCS and prestretch due to atrial contraction. Each data point represents a segment of the average healthy heart.