Transmural shear wave speed gradient distinguishes intrinsic myocardial stiffening from preload-induced changes in functional stiffness: an in silico study

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Introduction

Cardiac shear wave elastography (SWE) is a promising technique to differentiate healthy from pathological stiffer tissue based on shear wave propagation speed. However, shear wave propagation is sensitive to the operational stiffness of the myocardium which can alter by changes in intrinsic stiffness or by shifting along the stress-strain relation due to changes in pressure loading. Therefore, we built a finite element (FE) model of the left ventricle (LV) to investigate the sensitivity of SWE measurements to increased stiffness and pressure, with particular attention for transmural changes in shear wave speed.

Methods

A generic LV model with an isotropic and anisotropic Holzapfel-Ogden material law was considered in the FE software Abaqus (Abaqus Inc, Providence, RI, USA), as shown in fig. a and b. End-diastolic state was mimicked by applying a LV pressure of 8 mmHg and a right ventricular (RV) pressure of 4 mmHg. Then, the submodeling technique was used to isolate a smaller region with a refined mesh (local model in fig. a) for modeling SWE. Shear waves were simulated by applying a body force for 250 µs that mimics the acoustic radiation force associated with the push ultrasound beam. This workflow was repeated for a model with elevated LV pressure (20 mmHg) and a model with fibrotic material properties (fig. b). The transmural changes in shear wave speed and stiffness were analyzed for each simulation. Numerical findings were verified for the shear waves after mitral valve closure in a healthy volunteer, a patient with diastolic dysfunction (LV end-diastolic pressure of 24 mmHg) and a heart transplant patient with confirmed fibrosis.

Results

The shear wave propagation pattern is shown in fig. c for all three simulation cases using the isotropic material model in the parasternal long axis view (PLAX). As expected, the shear wave propagates faster for elevated pressure (2.5m/s) and fibrosis (3.1m/s) compared to the normal setting (1.4m/s) at mid-cardiac wall, as is also reflected by the changes in shear stiffness (20.7kPa and 10.4kPa vs. 3.0kPa). The anisotropic model showed only an increase in wave speed when pressure increased (3.5m/s vs. 0.7m/s at mid-cardiac wall), corresponding with shear stiffness changes (27.7kPa vs. 5.2kPa). The transmural analysis of the wave front showed a more curved front for elevated pressure than for fibrosis (fig. c), leading to higher transmural shear wave speed gradients for elevated pressures. This finding was consistent for both isotropic and anisotropic simulations (fig. d) and preliminary results in human subjects confirmed the elevated transmural shear wave speed gradient in the patient with elevated pressure (fig. d).

Discussion

Our data indicate that the transmural shear wave speed gradient, rather than the absolute shear wave speed, distinguishes changes in myocardial properties from changes in hemodynamic loading. While this study demonstrates the power of FE modeling of shear wave physics as a guide for post-processing in patients, confirmation of findings in a larger patient cohort is

warranted, as well as further attention for the anisotropic material model of myocardial fibrosis that was unable to generate the expected upturn in shear wave speed and shear stiffness.

Acknowledgements

References