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How do myocardial shear waves, measured with ultra-high frame rate echocardiography, differ between volunteers and patients with cardiac amyloidosis?

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Background: Myocardial stiffness (MS) is an important pathophysiological determinant of left ventricular (LV) dysfunction. Recently, cardiac shear wave (SW) elastography has been proposed as a new, non-invasive tool to assess MS. SW's occur after mechanical excitation of the myocardium, e.g. after mitral (MVC) and aortic valve closure (AVC), and their propagation velocity is directly related to MS, thus providing an opportunity to assess stiffness at end-diastole (ED) and end-systole (ES). Given the fast propagation of these waves and the relatively small size of the heart, imaging SW requires ultra-high frame rate echocardiography which is currently only available in experimental scanners.

Purpose: The aim of this study was to evaluate SW in healthy volunteers (HV) and patients with cardiac amyloidosis (CA) by using ultra-high frame rate ultrasound.

Methods: We prospectively included 31 HV (age 36 ± 12 years) and 14 patients with biopsy-proven CA (68 ± 8 years). Parasternal long axis views of the LV were acquired using a fully programmable experimental scanner (HD-PULSE) equipped with a clinical phased array transducer (Samsung Medison P2-5AC) at 1153 ± 162 frames per second. Both tissue (Doppler) velocity and acceleration maps were extracted from an anatomical M-mode line along the midline of the LV septum. The propagation velocity of SW occurring after AVC (ES) and MVC (ED) was measured as the slope in the M-mode image. Standard echocardiography using a commercially available scanner (Vivid 9, GE) was performed to determine diastolic function according to current guideline recommendations (grade 1 to 3).

Results: SW propagation velocity differed significantly between HV and CA, both after MVC (3.41 ± 0.85 vs. 6.86 ± 2.35 m/s; p < 0.01) and AVC (3.53 ± 0.71 vs. 5.61 ± 1.12 m/s; p < 0.01; Figure 1A). SW propagation velocity after MVC was found to be significantly different among the group with normal diastolic function and groups with diastolic dysfunction (ANOVA, p < 0.01; Figure 1B). Positive correlation was found between SV velocity after MVC and E/E’ as a conventional echocardiographic parameter of diastolic function (r = 0.87, p < 0.001; Figure 1C).

Conclusions: Both, end-systolic and end-diastolic shear wave velocities were significantly higher in patients with cardiac amyloidosis than in healthy volunteers, indicating a higher stiffness of the myocardium in cardiac amyloidosis. End-diastolic shear waves showed a good correlation with traditional parameters of diastolic function. Our data suggest, that cardiac shear wave elastography has the potential to become a powerful tool for the assessment of myocardial properties.
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Figure 1: SW velocity propagation in healthy volunteers and patients with amyloidosis. A) Comparison of SW propagation velocity after MVC and AVC between HV (n = 31) and patients with CA (n = 14); B) SW propagation velocity after MVC in subjects with different grade of diastolic dysfunction (DD); C) Correlation between SW propagation velocity after MVC and conventional echo parameter of diastolic function (E/E').