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Poster presentation

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CMR characterization of the septal bounce in patients with constrictive pericarditis

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Introduction

The observation of a 'septal bounce' is frequently helpful in diagnosing constrictive pericarditis (CP). The mechanism behind this phenomenon is unknown.

Purpose

Using advanced CMR, we hypothesized that the etiology of the septal bounce in CP is a result of the differential AV-valves inflow patterns in relation to the ventricular septum, due to realignment of the tricuspid valve plane.

Methods

We compared SSFP 4-chamber view images (TR = 25-35 ms) between 11 CP patients and 11 controls. We deconvoluted the mechanics of the septal bounce via SSFP, phase-velocity mapping (PVM) and Quiver-plots, and correlated it with the diastolic phases of the cardiac cycle. We then related the mechanisms behind this time-correlation using three metric measurements at end-systole: "tri-septal angle" between tricuspid annulus and proximal ventricular septum (Fig. 1), "tricuspid annulus angulation ratio" (TAAR) as defined in Fig. 2, and "right ventricular clamp" (RV clamp) ratio as {(cardiac area-RV area)/cardiac area}.

Results

The observed septal bounce in CP is composed of two sequential movements, each consisting of an impingement of the septum from the RV towards the LV followed by recovery of this deformation. The initial movement, of larger amplitude, occurs at 126 ± 57 ms following the opening of the mitral valve, while the second occurs at 39 ± 39 ms after the initiation of atrial systole. The tri-septal angle was 81° ± 9° in CP versus 91° ± 7° in controls (p = 0.01). This is explained by the fact that the insertion of the tricuspid annulus on the RV free wall at end-systole is more distal in CP: TAAR was 1.10 ± 0.05 in CP vs. 0.98 ± 0.06 in controls (p = 0.001). Quiver-plots of PVM velocities in Fig. 3 illustrate the tricuspid and mitral inflows. The RV clamp was higher in CP (0.88 ± 0.03) versus controls (0.85 ± 0.03, p = 0.02).

Conclusion

The two movements of the septal bounce are determined by cardiac mechanisms that we defined using time-correlations in diastole and CMR metrics. As demonstrated by tri-septal angles and Quiver-plots, in patients with CMR-confirmed CP the brisk high-velocity tricuspid inflow strikes the ventricular septum at a more acute angle, while the mitral inflow runs parallel to the septum, resulting in two distinct septal movements. In controls, inflow patterns are parallel to the septum. We suspect that this is due to the observation that in CP, at end-systole, the cardiac chambers tend to 'clamp' the RV and aggregate towards the RV outflow (the latter possibly behaving as a pressure escape). Thus, CMR appears to identify the elusive mechanism of the septal bounce.

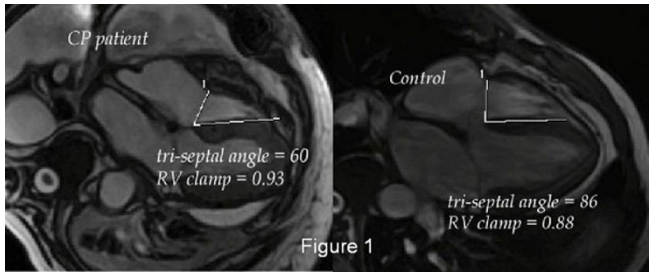

Figure 1

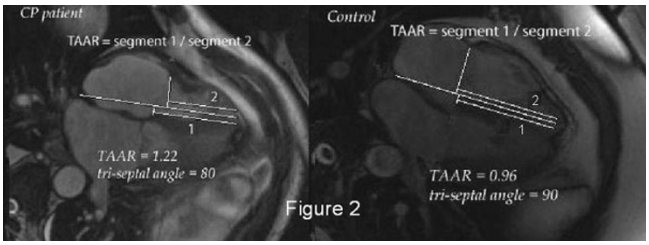

Figure 2

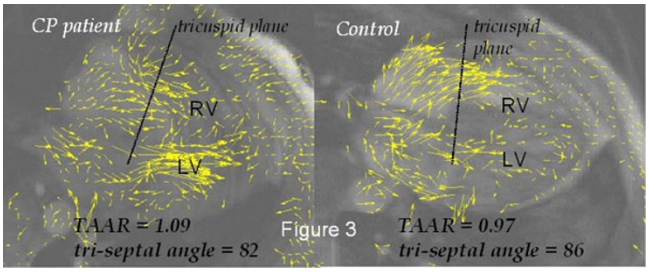

Figure 3