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Principle component analysis of myocardial strain to quantify left ventricular dyssynchrony

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Background

Cardiac resynchronization therapy (CRT) is effective for selected heart failure (HF) patients, but is associated with a 30-40% nonresponse rate. Identification of CRT responders may be improved with myocardial strain imaging. The circumferential uniformity ratio estimate (CURE)[1] measures mechanical dyssynchrony by Fourier series fitting of myocardial strains over space, but requires user interaction to define a range of cardiac phases over which CURE is calculated (time dependence). We hypothesize that principal component analysis (PCA) can quantify dyssynchrony in myocardial strain in a data-driven, time-independent manner that does not require any subjective user assessments of strain data.

Methods

Dyssynchronous HF was induced in canines (N=5) with tachycardia pacing and left bundle branch ablation (LBBB-HF), while synchronous HF with narrow QRS (NQRS-HF) was induced in canines with tachycardia pacing only (N=5). Four normal canines were also studied.

Spiral cine DENSE MRI was performed on a 1.5 T scanner (Avanto, Siemens) in all 14 canines. 2D myocardial motion was tracked in a mid-ventricular short-axis plane from DENSE images acquired using the following parameters[2]: interleaves=6, TR=17 ms, TE=1.9 ms, slice thickness=8 mm, excitation flip angle=15°, in-plane resolution=2.8 x2.8 mm and displacement-encoding frequency= 0.1 cycles/mm.

DENSE images were analyzed[3] to calculate left ventricular (LV) circumferential strain (Ecc), and PCA was applied to the Ecc-time curves for a 24-segment LV model. Specifically, the LV Ecc curves were decomposed spatially into principal component basis vectors. The PCA-based metric for measuring LV dyssynchrony, termed First Order Regional Conformity Estimate (FORCE), was calculated as $|\text{sum}(PCL_1)|/\text{sum}(|PCL_1|)$ where PCL_1 represents the loadings of the first principal component basis vector. Both FORCE and CURE range from 0 (dyssynchrony) to 1 (perfect synchrony). The Kruskal-Wallis one-way ANOVA test was used to compare FORCE and CURE among the three groups in pairwise fashion.

Results

Figure 1 shows the spatial distribution of PCL_1 for the Ecc of example LBBB-HF and NQRS-HF canines. The PCL_1 of LV Ecc in LBBB-HF canines varied widely over LV segments, whereas PCL_1 showed little variation over LV segments in NQRS-HF and normal canines. As shown in Figure 2, FORCE and CURE were both markedly different in LBBB-HF versus NQRS-HF ($p<0.05$) and LBBB-HF versus normal ($p<0.05$). Also, FORCE was significantly greater than CURE for NQRS-HF ($p<0.05$), indicating even better identification of synchrony than CURE.

Conclusions

PCA, using the promising new FORCE parameter, effectively and automatically identifies mechanical dyssynchrony in HF in a data-driven and completely time-independent fashion. Further clinical evaluation of FORCE for prediction of CRT response is warranted.

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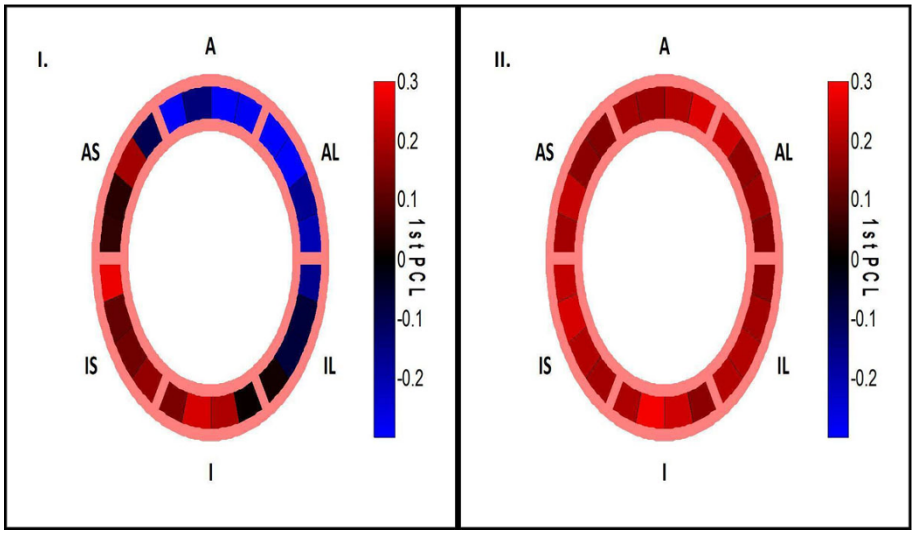

Figure 1 Bulls-eye plots of PCL_1 as a function of LV segments for I) a LBBB-HF and II) a NQRS-HF canine; A-Anterior, AL-Anterolateral, IL-Inferolateral, I-Inferior, IS-Inferoseptal, AS-Anteroseptal.

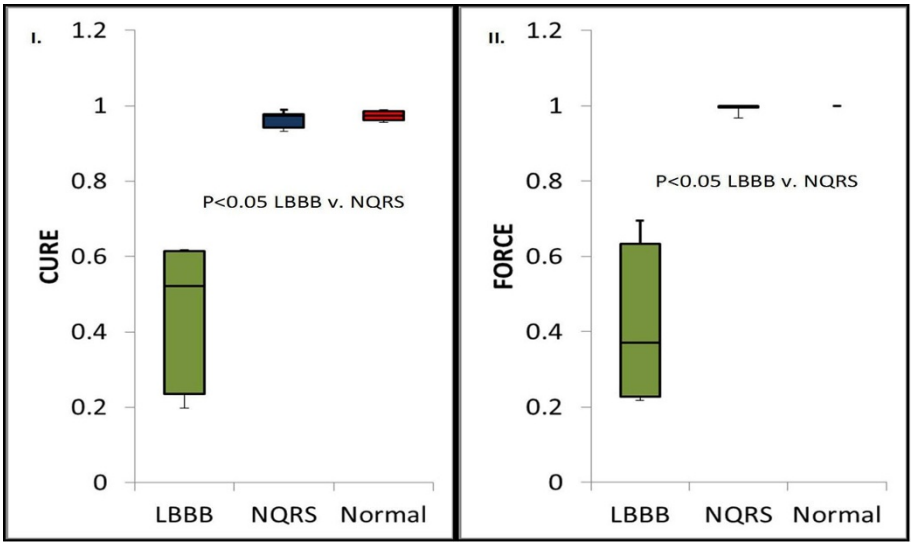

Figure 2 Boxplot comparisons of I) CURE and II) FORCE for LBBB-HF,NQRS-HF and Normal dogs.

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