ACC.14
TCT@ACC-i2 | innovation in intervention

A924
JACC April 1, 2014
Volume 63, Issue 12

Heart Failure and Cardiomyopathies

THE RELATIONSHIP BETWEEN RADIAL STRAIN DELAY INDEX BASED ON BOTH SEGMENTAL TIMING AND AMPLITUDE AND HAEMODYNAMIC RESPONSE TO CARDIAC RESYNCHRONIZATION THERAPY

Poster Contributions
Hall C
Monday, March 31, 2014, 9:45 a.m.-10:30 a.m.

Session Title: Heart Failure and Cardiomyopathies: Therapy V
Abstract Category: 14. Heart Failure and Cardiomyopathies: Therapy
Presentation Number: 1260-161

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Background: Correcting mechanical dyssynchrony is proposed as one of the major mechanisms of benefit from CRT. Very early and late segmental contraction may not fully contribute to end-systolic function. A novel radial strain delay index (RSDI) can be utilised to quantify reserve of wasted contraction, caused by dyssynchrony and segments of low amplitude strain or scar, prior to CRT. We hypothesized that a greater extent of wasted contraction prior to CRT would relate to a greater haemodynamic response.

Methods: We examined data from 116 patients undergoing CRT with an optimally placed LV lead (concordant or adjacent to latest site of activation). Echocardiograpy was perfomed prior to implantation and at 6 months follow-up. RSDI was calculated using speckle tracking radial strain as the sum of the difference between peak and end-systolic strain (%) across 12 (basal and mid) LV segments. >15% reduction in LVESV defined CRT responders. Within 24 hours of CRT implant non-invasive cardiac output (CO) monitoring based on bioreactance cardiography was performed with the device switched off (baseline) and with the device turned on (AV delay 120ms, VV delay OMS).

Results: Out of 116 patients (QRS 157±22, EF 24±6.3, 52% ischemic etiology) 70% were classified as CRT responders. RSDI was higher at baseline in responders than non-responders (67±36.5% vs. 36±26%, p40 (previously defined as optimal cut-off to predict CRT response) had a greater increase in CO when compared to those patients with RSDI 13%±14 vs. 5%±18 <40, p=0.009.

Conclusion: In patients with an optimally placed LV lead higher RSDI appears to translate into greater improvement in cardiac output. This may reflect a greater potential for recruitment of wasted energy as a therapeutic target for CRT.