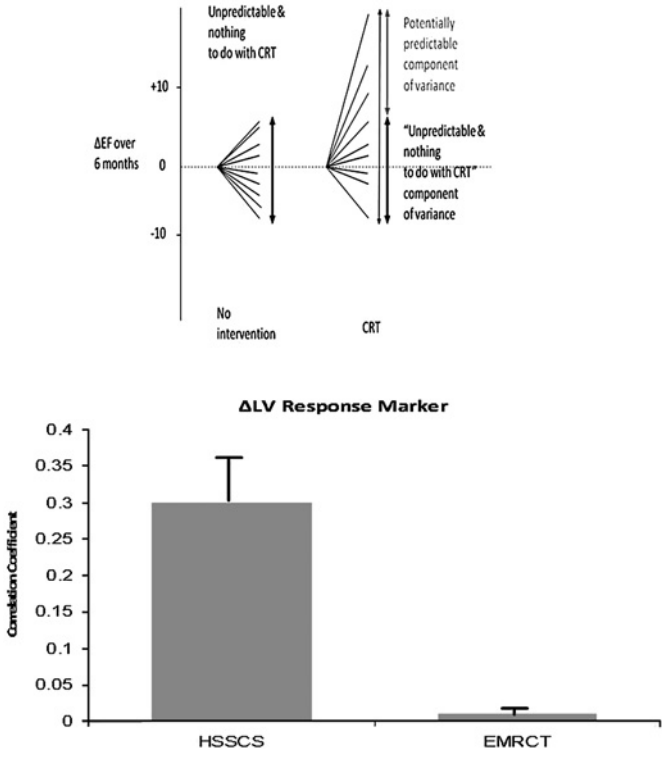
dyssynchrony and remodelling response in contrast to EMRCTs ($p<0.0000000001$), whether response is LVEF (0.40 vs 0.01), ESV (0.26 vs 0.01); EDV (0.53 v 0.01). An "averaged" reported r^2 between differing dyssynchrony markers to commonly used echocardiographic response markers is shown in Abstract 85 figure 1, lower panel.



Abstract 85 Figure 1

EMRCT data shows maximal r^2 between dyssynchrony and ΔLVEF is 0.57 (ΔESV, 0.54; ΔEDV, 0.50). Dyssynchrony indices' own variability further contracts observable r^2 values (by x0.68). The overall ceiling to r^2 is between dyssynchrony and ΔLVEF is 0.39 (ΔESV, 0.37; ΔEDV, 0.34). All EMRCT r^2 values obey these statistical limits; 29% of HSSCSs results do not.

Conclusions HSSCSs suggest dyssynchrony markers strongly predict response to BVP but EMRCTs cannot confirm this. Natural variability forces observed correlation coefficients between dyssynchrony and response to be low. EMRCTs, being less susceptible to publication bias, reflect this reliably. Frequent citation (without verification in independent cohorts) of the most exuberant values, from HSSCSs creates mathematically unviable, unrealistic, expectations. Simply searching for progressively more extreme correlations is therefore misguided. Rationally, we should concentrate on improving test-retest reproducibility of markers of dyssynchrony and of response.

86 HOW OFTEN IS IMPORTANT ADJUSTMENT OF PACING INTERVALS REQUIRED FOR OPTIMAL RESPONSE FOLLOWING CRT?

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Introduction A significant minority of patients do not experience clinical benefit following cardiac resynchronisation therapy (CRT). Haemodynamically-guided adjustment of the intervals between chambers paced ("optimisation" of atrio-ventricular (AV) and left-right ventricular (VV) delays) may be undertaken to improve the chance of response to CRT. However, data to support this approach as standard management are lacking and many institutions programme CRT devices to deliver "out-of-the-box" intervals, only undertaking optimisation when clinical response is lacking. We sought to determine how often the "out-of-the-box" settings are optimal or acceptable and how often CRT optimisation results in significant alteration of the pre-programmed pacing intervals.

Methods Data were collected from 180 consecutive patients who underwent CRT followed by optimisation within 24 h. Optimisation was performed with serial adjustment of AV and VV intervals. Haemodynamic assessment was undertaken using either echocardiography or Non-Invasive Cardiac Output Measurement. The optimal pacing intervals were considered to be those which resulted in greatest acute augmentation of cardiac output and the device was programmed accordingly. The final settings were compared with the pre-programmed settings for that device and the difference (AV or VV Adjustment) derived, taking into account the preset paced or sensed AV delay. An AV or VV Adjustment of more than 40 ms was considered to be clinically significant. Data are presented as mean (SD).

Results Optimal AV delay ranged from 60 to 200 ms (mean 124 ms (30)), VV delay ranged from 0 to 100 ms (mean 23 ms (19)). With the pre-set pacing parameters, cardiac output was acutely augmented by 13.1 (34)%. Optimised CRT produced further improvement of cardiac output, to 24.9 (32)% augmentation. "Out-of-the-box" settings were found to be optimal in 11 (6.1%), or requiring only minor alteration in 120 (66.7%). A clinically significant alteration in AV delay was made in 40 (22.2%), in VV delay in 12 (6.7%) or in either parameter in 49 (27.2%).

Conclusions Significant adjustment of AV or VV delay is required in over a quarter of patients receiving CRT. Optimisation of pacing intervals provides augmentation of cardiac output over and above the "out-of-the-box" settings. The findings suggest that optimisation is an important component of resynchronisation therapy.

Abstract 86 Table 1 Adjustment of pacing intervals following optimisation of CRT

	0	1–20 mS	21–40 mS	41–60 mS	61–80 mS	81–100 mS
AV Adjustment	29 (16.1)	89 (49.4)	22 (12.2)	32 (17.8)	7 (3.9)	1 (0.6)
VV Adjustment	50 (27.6)	65 (35.9)	53 (29.3)	11 (6.1)	0	1 (0.6)

Data as N (%).

87 OPTIMISATION OF VV DELAY OF CRT IS MORE REPRODUCIBLE USING PEAK VELOCITIES THAN USING VELOCITY TIME INTEGRAL, AS WELL AS BEING QUICKER

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Background It is not obvious which is a better echocardiographic marker for optimisation of AV or VV delay: stroke distance (VTI) or peak velocity. The biggest problem is genuine physiological variability between beats. Because optimisation of VV delay requires detection of persistent changes in cardiac function ("signal"), which may be small in relation to beat-to-beat variability ("noise"), we should choose measurements with the best signal-to-noise ratio and reproducibility. The standard echocardiographic method of choice for VV delay optimisation is to maximise left ventricular outflow tract velocity time integral (LVOT VTI). An alternative is peak velocity instead of VTI as the parameter to be measured. But surely VTI, which is encompassing and cumulating more data, is more immune to disruption by spontaneous variability between beats,