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 vs 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**: Data shows maximal $r^2$ between dyssynchrony and $\Delta$LVEF is 0.57 ($\Delta$ESV, 0.54; $\Delta$EDV, 0.50). Dyssynchrony indices’ own variability further contracts observable $r^2$ values (by $0.68$). The overall ceiling to $r^2$ is between dyssynchrony and $\Delta$LVEF is 0.59 ($\Delta$ESV, 0.57; $\Delta$EDV, 0.54). 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.

**Table 1**: Adjustment of pacing intervals following optimisation of CRT

<table>
<thead>
<tr>
<th>Data as N (%)</th>
<th>0</th>
<th>1–20 mS</th>
<th>21–40 mS</th>
<th>41–60 mS</th>
<th>61–80 mS</th>
<th>81–100 mS</th>
</tr>
</thead>
<tbody>
<tr>
<td>AV Adjustment</td>
<td>29 (16.1)</td>
<td>89 (49.4)</td>
<td>22 (12.2)</td>
<td>37 (17.8)</td>
<td>7 (3.9)</td>
<td>1 (0.6)</td>
</tr>
<tr>
<td>VV Adjustment</td>
<td>50 (27.6)</td>
<td>65 (35.9)</td>
<td>53 (29.3)</td>
<td>11 (6.1)</td>
<td>0</td>
<td>1 (0.6)</td>
</tr>
</tbody>
</table>

**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,
and therefore simply using peak velocity might give a less reliable optimum? Surely the time saved by using peak would have a price to pay in poorer reproducibility of the optimum? In this study, we evaluate whether peak velocity is a suitable alternative to VTI, having regard to both time consumed and reproducibility. We also examine whether averaging multiple replicate measurements improves optimisation.

**Methods & Results** VV optimisation was performed on 40 subjects with biventricular pacemakers using LVOT velocity (VTI or peak) as the echocardiographic marker being maximised. Importantly, 6 successive replicate optimisations were performed per patient at a single session. Scatter of apparent VV optimum between repeat optimisations were performed per patient at a single session. Peak velocity had a higher intraclass correlation coefficient (ICC) than VTI (0.66 vs 0.55, p=0.005). Scatter between replicate optimisations is reduced if, instead of single measurements, we use pairs, or triplicates (ANOVA p<0.0001). This benefit occurs with both peak and VTI (p<0.001 among each). Time taken for acquisition and analysis of a single optimisation (6 settings) was 17.5 s for peak and 57.5 s for VTI (p<0.0001).

**Conclusions** Doppler optimisation of VV delay using peak velocity rather than VTI is (as expected) quicker but (surprisingly) more accurate. Making replicate measurements further improves reproducibility. Perhaps guidelines should favour peak over VTI and mandate multi-replicate averaging? These data suggest a rare opportunity to reduce labour while increasing reliability of optimisation. Indeed, triplicate peak velocity assessment takes the same amount of time as a single VTI, and identifies the VV optimum 3 times more confidently. While VTI measurement remains essential for assessing stroke volume and cardiac output, for optimisation purposes it comparison of peak velocity between different settings is both faster and more reliable.

**Abstract 87 Figure 1**

**EVALUATION OF THE IMPACT OF AV DELAY VARIATION ON THE ACUTE MECHANOCENERGETIC EFFICIENCY OF CARDIAC RESYNCHRONISATION THERAPY AND ASSESSMENT OF PERFORMANCE OF NON-INVASIVE VS INVASIVE HAEMODYNAMIC OPTIMISATION**

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**Background** The impact of varying AV delay on the acute mechanoe energetic efficiency of cardiac resynchronisation therapy (CRT) is not known; nor is known if non-invasive haemodynamic optimisation by blood pressure agrees with invasive haemodynamic measures during optimisation. We studied these invasively, in contemporary patients.

**Methods** Eleven patients with heart failure (EF 29±8%) and left bundle branch block (LBBB, QRS 154±26 ms) underwent measurements of left ventricular (LV) pulse pressure (systolic minus diastolic), aortic flow velocity and myocardial oxygen consumption (MVO2) at four settings: 3 AV delays during biventricular (BiV) pacing (reference BiV-AV120 ms; BiV-AV40 ms; individualised haemodynamic BiV-AVoptimum), and at intrinsic ventricular conduction (LBBB). Atrial pacing at 100 bpm ensured a fixed heart rate.

**Results** LV pulse pressure rose from LBBB to BiV-AV120 ms by 10±2% (p<0.001) and 2±1% more (p<0.05) at the haemodynamic BiV-AVoptimum. At BiV-AV40 ms, pressure was 10±2% worse than BiV-AV120 ms (p<0.001), no different to LBBB (Δ=0.8±0.4%, p=ns). Invasive aortic flow velocity, measured at a fixed position throughout each individual’s study (ie, cardiac output index), rose by 9±2% (p<0.01) from LBBB to BiV-AV120 ms, rising a further 5±1% (p<0.01) at BiV-AVoptimum. At BiV-AV40 ms, aortic flow was, no different to LBBB (p=NS). MVO2 increased from LBBB to BiV-AV120 ms by 9±4% (p=0.005) and to BiV-AVoptimum by 12±3% (p=0.002). MVO2 at BiV-AV40 ms and LBBB was not significantly different (Δ4±3%, p=ns). The 4 pacing states lay on a straight line: for Δpressure against Δflow, r=0.99 (p<0.01), Abstract 88 figure 1. External work (Δpressure X Δflow) correlated with Δ MVO2, r=0.99 (p<0.01), with slope 1.61±0.17, significantly greater than 1.00 (p<0.05), Abstract 88 figure 2.

**Abstract 88 Figure 1** The correlation of LV pulse pressure and aortic flow velocity during acute biventricular pacing, (at three AV delays) and during LBBB, at a fixed heart rate.

**Abstract 88 Figure 2** The correlation of cardiac work and myocardial oxygen consumption during acute biventricular pacing, (at three AV delays) and during LBBB, at a fixed heart rate.