choice has changed in favour of rotary pumps; 19%, 69% and 96% for E1, E2 and E3 respectively. Median duration of VAD support increased from 84 days (IQR 20–209) in E1 to 280 days (IQR 86–661) in E3 (p<0.01). Overall survival to 1 year after VAD implant rose from 52.9% (95%CI 40 to 64) in E1 to 65.6% (95%CI 54 to 75) in E3 (p=0.10). Of the 239 patients implanted, 83 (35%) have undergone HTx, 52 (22%) are alive on VAD support & 84 (35%) died on support. Twenty were explanted following myocardial recovery; 18 of these remain alive & 2 died. Survival after HTx for patients with or without a pre-HTx VAD was 81.4% (95%CI 71 to 88) & 90.3% (95%CI 88 to 92) respectively at 30-days (p<0.01) and 80.0% (95%CI 63 to 82) & 84.3% (95%CI 82 to 87) respectively at 1-year (p<0.01). 1-year survival conditional on 30-day survival was similar with & without a pre-HTx VAD (93% vs 91%, p=0.48).

Conclusion Heart transplant activity has declined and waiting times have become prolonged leading to an increased need for bridging to transplantation. There has been a shift from volume displacement VADs to rotary blood pumps and the duration of support has increased. Post VAD survival has improved. While bridging appears to increase mortality early after HTx, longer term survival is unaffected.

83 CLINICAL AND HAEMODYNAMIC STATUS BEYOND 3 MONTHS OF MECHANICAL SUPPORT WITH THE HEARTWARE VENTRICULAR ASSIST DEVICE

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Introduction Limited data exist on the longer term clinical and haemodynamic impact of the HeartWare left ventricular assist device (HVAD®) when used as a bridge to heart transplantation. Patients who had a device longer than 3 months were reviewed.

Methods 26 patients had a HVAD implanted from 07/2009 to 07/2010 (mean age 46.8 years, 18 male, 5174 total days of support). Baseline and follow-up NYHA functional class, peak VO2 (bicycle exercise), right heart haemodynamics, biochemistry and mortality outcome were compared using paired t test. Results: 22/26 (85%) patients survived beyond 3 months. 4 patients died before (mean survival 40 days, 2 stroke and 2 multi-organ failure) and 2 died after (mean survival 173 days, 1 stroke, 1 right heart failure) discharge from hospital. 2 patients were transplanted (at 3 and 241 days after implant) and 1 had recovery of LV function. Follow-up data is available for 14/20 survivors (mean 197 days from implant). Significant results are shown in the Abstract 83 table 1. There was no significant change in peak VO_2 (9.9±1.8 to 12.9±3.8, p=0.08), haemoglobin (12.7±1.7 to 12.1±1.2, p=0.3) or creatinine (122±41 to 105±38, p=0.19).

Abstract 83 Table 1

Parameter	Baseline	Follow-up	p Value
NYHA functional class	3.6±0.4	2.1±0.6	<0.001
Mean PA pressure (mm Hg)	38±9	21±8	<0.001
Mean PW pressure (mm Hg)	25±5	10±5	<0.001
Transpulmonary gradient (mm Hg)	12±5	9±3	0.02
Right atrial pressure (mm Hg)	11±6	5±4	0.006
PA oxygen saturation (%)	51±8	66±7	0.003
Cardiac Output (l/min)	2.9±0.8	4.3±0.9	<0.001
Sodium (mmol/l)	134±4	139±3	0.002

Conclusions The HVAD® results in significant improvement in functional class, right heart haemodynamics, cardiac output and sodium levels beyond 3 months of therapy. Ongoing randomised clinical trials will establish the long-term outcome of this device.

84 TREATMENT OF REFRACTORY RIGHT HEART FAILURE AFTER IMPLANTATION OF A LEFT VENTRICULAR ASSIST DEVICE. IS THE LEVITRONIX CENTRIMAG RIGHT HEART SUPPORT A SOLUTION?

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Introduction Right heart failure after left Ventricular Assist Device (LVAD) implantation is a severe complication, in extreme cases necessitating additional mechanical assist. We present our institutional experience with the Levitronix CentriMag used for right ventricular support commencing LVAD implantation with refractory right ventricular failure.

Material and Methods Between March 2001 and November 2010 109 patients underwent implantation of long term, total implantable, continuous flow LVADs: 60 HeartMate II, 25 Jarvik 2000 and 24 HeartWare. All patients requiring right ventricular support were included (n=24), for which the Levitronix CentriMag continuous flow, paracorporeal device was used. The analysis included patient demographics as well as overall duration of support and outcome parameters, including survival at 30, 90 days and 1 year.

Results 24 pts. underwent implantation, age 37.9±13.7 years, gender: M/F-15/9, underlying disease: dilated cardiomyopathy 22 (92%), peripartum cardiomyopathy 1(4%), viral myocarditis 1(4%). Median duration of support: 28 days (5–146). 3(12.5%) pts. underwent heart transplantation (HTx) on RV support, 14(58.5%) underwent RVAD explantation. Of these, 3 underwent successful HTx, 4 recovered LV function and underwent successful LVAD explantation, 3 remain on continuing LVAD support, 4 patients died after RVAD explantation (post explantation day 1, months 3 and 4 and at 2 years), 7(29%) patients died during RV support. Median ITU/hospital stay: 19.5 days (6–145)/78.5 days (10–219). 30-day/90-day/1-year survival: 79%/71%/60%. 15(62.5%) patients were discharged from hospital after treatment. Median survival after procedure: 473.5 days (10–1917).

Conclusion Levitronix CentriMag right ventricular support is an excellent option for post LVAD implantation treatment of refractory RV failure. It allows either bridging to transplantation or RV function improvement and provides an acceptable rate of survival.

85 PREDICTION OF RESPONSE TO BIVENTRICULAR PACING FROM DYSSYNCHRONY INDICES: THE ABSOLUTE LIMIT ON PREDICTABILITY, AND ITS CLINICAL IMPLICATIONS

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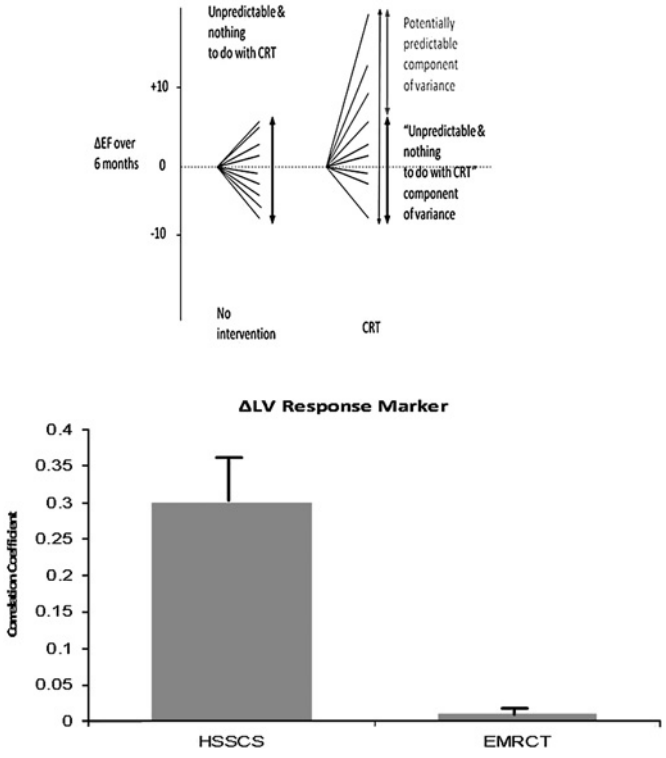
Background It may be incorrect to believe that, with a good echocardiographic marker of mechanical dyssynchrony, response to biventricular pacing (BVP) should be predictable with a high r^2 value. Variability between repeat echocardiographic measurements, and between successive dyssynchrony measurements, may reduce r^2. Both will mandatorily limit the achievable r^2; we determine this "contraction factor".

Method and Results We compared correlation coefficients of dyssynchrony indices with response markers, in externally monitored randomised controlled trials (EMRCTs) and highly skilled single centre studies (HSSCSs). ΔLVEF in CRT recipients comprises true CRT effect plus unpredictable spontaneous variability present in control patients (Abstract 85 figure 1, upper panel). The resultant depression in r^2 is calculated. HSSCSs overstate r^2 between

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,