majority of the increase in coronary flow occurred in diastole (64±8%, p=0.03). The systolic compression waves also increased, forward by 33±11% (p=0.043) and backward by 33±10% (p=0.014). BiV-120 generated a smaller LV dP/dt_{max} (by 23±12%, p=0.034) and $negdP/dt_{max}$ (by 23±10%, p=0.039) increase than BiV-OPT, against LBBB as reference; BiV-Opt and BiV-120 were not statistically different in coronary flow or waves. BiV-40 was no different from LBBB.

Conclusions When biventricular pacing improves left ventricular contractility, it increases coronary blood flow, predominantly by increasing the dominant diastolic backward decompression (suction) wave.

007 SHOULD CURRENT MODALITIES OF VV OPTIMISATION BE TRUSTED? AN ASSESSMENT OF THE INTERNAL VALIDITY OF ECHOCARDIOGRAPHIC, ELECTROCARDIOGRAPHIC AND HAEMODYNAMIC MODALITIES OF OPTIMISATION

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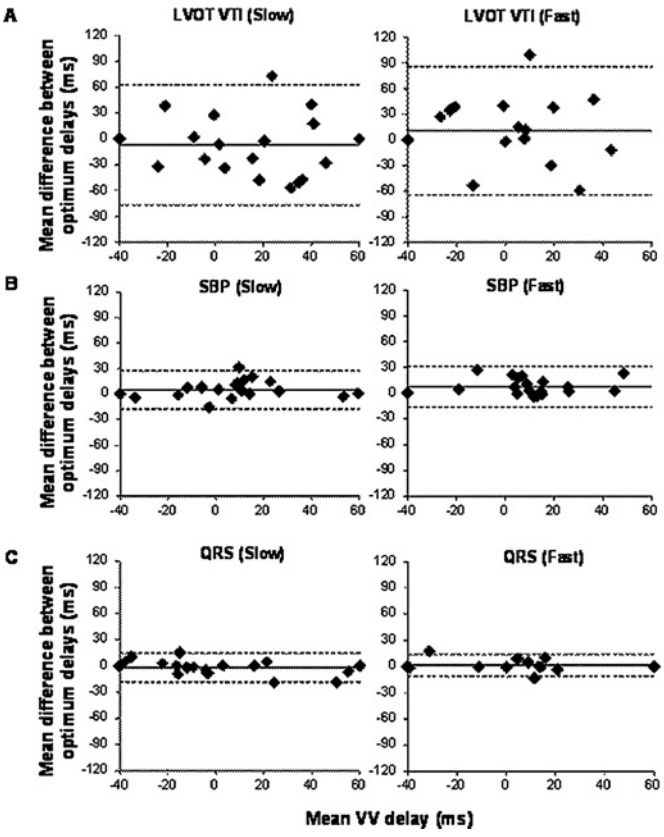
Background In atrial fibrillation (AF), VV optimisation of biventricular pacemakers can be examined in isolation. We used this approach to evaluate the internal validity of three VV optimisation methods.



Abstract 007 Figure 1 The reproducibility (SD of the difference, SDD in ms) of LVOT VTI (A) is equally poor at the slow (31 ms) and fast (35 ms) heart rates. SBP reproducibility (B) was better than LVOT VTI at slow (10 ms, p<0.01) and fast heart rates (9 ms, p<0.01). The reproducibility of the QRS width (C) was also better at slow (8 ms, p<0.01) and fast (6 ms, p<0.01) rates.

Methods 20 patients (16 men, age 75±7) in AF were optimised, at two paced heart rates, by LVOT VTI (flow), non-invasive arterial pressure, and ECG (minimising QRS duration). Each optimisation method was evaluated by: singularity (unique peak of function), reproducibility of optimum, and biological plausibility of the distribution of optima.

Results The reproducibility (SD of the difference, SDD) of the optimal VV delay is 10 ms for pressure, vs 8 ms (p=NS) for QRS and 34 ms (p<0.01) for flow. Singularity of optimum is 85% for pressure, 63% for ECG and 45% for flow (χ^2=10.9, p<0.005). The distribution of pressure optima is biologically plausible, with 80% LV pre-excited (p=0.007). The distributions of ECG (55% LV pre-excitation) and flow (45% LV pre-excitation) optima are no better than random (p=NS). The pressure-derived optimal VV delay is unaffected by the paced rate: SDD between slow and fast heart rate is 9 ms, no different from the reproducibility SDD at both heart rates, (p=NS).

Conclusions Using non-invasive arterial pressure, VV delay optimisation is achievable with good precision, satisfying all 3 criteria of internal validity, and the optimum is unaffected by heart rate. Neither QRS minimisation nor LVOT VTI satisfy all validity criteria and are therefore weaker candidate modalities for VV optimisation.

008 LEFT VENTRICULAR HYPERTROPHY IS ASSOCIATED WITH INTRAMURAL DYSSYNCHRONY ON EXERCISE IN PATIENTS WITH HEART FAILURE AND NORMAL EJECTION FRACTION

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Background The pathophysiology of heart failure with normal ejection fraction (HFNEF) is complex and not fully understood. We hypothesised that left ventricular hypertrophy (LVH) which is found in most patients with HFNEF might lead to intramural dyssynchrony and uncoupling of the complex 3-dimensional motion of the left ventricle (LV) particularly on exercise.

Method 33 patients with the clinical diagnosis of HFNEF (age 69±11 years, 19 female, EF 60±7%) and LVH (according to American Society of Cardiology, female >95 g/m^2, male 115 g/m^2) underwent detailed 2D-echocardiography at rest and on supine exercise. They were compared to 41 clinically diagnosed HFNEF patients without LVH (age 73±8 years, 26 females, EF 61±7%) and 35 age-matched control subjects (age 71±7 years, 27 females, EF 63±7). All subjects underwent cardiopulmonary exercise test to assess peak oxygen consumption (peak VO2). Echocardiographic images were analysed off-line. Apical and basal rotation and radial displacement were measured by speckle tracking. Longitudinal displacement was assessed by colour tissue Doppler imaging. Raw data and timing of events were analysed using a custom-written interpolation algorithm. SDSM (SD of four LV peak systolic motions: basal and apical rotation, longitudinal and radial displacement) was calculated.

Results SDSM was comparable at rest for all three groups but controls showed the highest reduction in SDSM compared to both groups of patients on exercise. Patients with LVH had the smallest reduction in SDSM implying greater dyssynchrony in LV motions on exercise. SDSM on exercise correlated with left ventricular mass index (r=0.362, p=0.002) and VO_2max (r=−0.319, p=0.011).

Conclusion LVH in patients with HFNEF is associated with intramural dyssynchrony and uncoupling of the complex 3-dimensional LV motions on exercise. This might contribute to their exertional symptoms.

Abstract 008 Table 1

	HFNEF with LVH	HFNEF without LVH	Controls	p-value (ANOVA)
SD systolic motions (ms) at Rest	53.3±32.7	45.5±33.2	44.8±25.7	0.456
SD systolic motions (ms) on exercise	48.0±28.3* †	28.7±18.7	25.7±15.7	<0.001

*p<0.05 compared to controls.
†p<0.05 compared to HFNEF patients without LVH.

009 UNCOVERING THE MECHANISM OF THE PARADOXICAL ASSOCIATION BETWEEN CARDIAC DYSSYNCHRONY AND BETTER SURVIVAL IN HEART FAILURE

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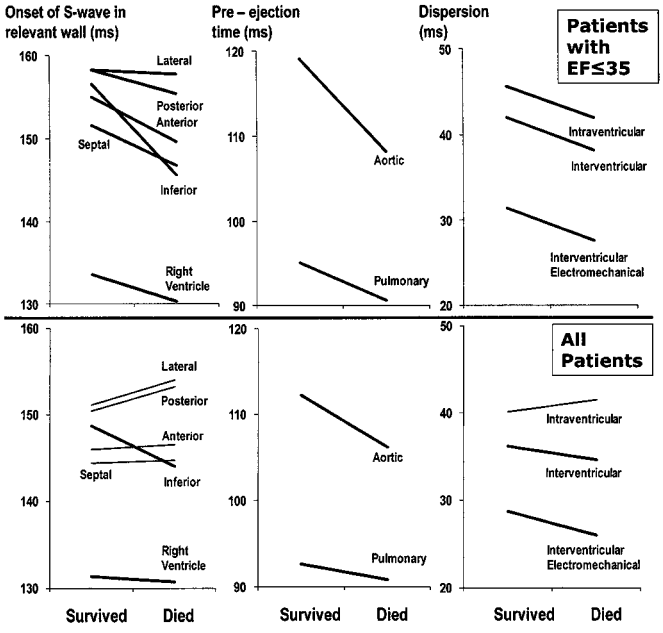
Introduction Paradoxically, dyssynchrony before CRT is associated with a better prognosis. We tested whether this was dependent on device implantation or on how the cohort was defined (EF ≤35 vs All-comers).
Methods 419 patients (67.8±11.3 years, 79.2% males, 127 deaths) with heart failure had echocardiographic assessment of mechanical dyssynchrony and were followed up (median 3.1 years).
Results 135 had dyssynchrony and 62 received CRT. The mean EF was 33.1±15.0%; 157 (35.2%) had an EF >35%. Among patients with EF≤35% (n=249), shorter aortic pre-ejection time (ie, *less* dyssynchrony) was associated with a worse prognosis (p<0.05). All dyssynchrony markers were higher in survivors (p<0.001 by sign test, upper panel). EF was not prognostic and depressed by dyssynchrony (r=−0.4, p<0.001). By examining all patients (regardless of EF); the association between dyssynchrony and better survival disappeared (p>0.05, lower panel). EF was restored to its prognostic significance (p=0.02). Taking a different approach to define poor ventricular function—using low S-wave velocity—EF had prognostic significance (p<0.05) and dyssynchrony markers were non-prognostic (p>0.05).
Conclusion Dyssynchrony predicts better survival in low EF groups because dyssynchrony artifactually lowers EF without damaging survival. The effect is independent of CRT. Replacement of EF with dyssynchrony-neutral measures of LV function, for example, peak S-wave velocity would avoid the appearance that dyssynchrony is favourable.



Abstract 009 Figure 1

010 MULTICENTRE VALIDATION OF THE ADVERSE PROGNOSTIC IMPLICATIONS OF DECLINING SERUM ALBUMIN LEVELS IN CHRONIC HEART FAILURE

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Background Single-centre studies have shown that a low serum albumin at baseline forecasts enhanced mortality in chronic heart failure (CHF) possibly because it reflects aberrations (eg, inflammation, impaired nutrition, plasma volume expansion) that can exacerbate disease. We hypothesised that attenuations in serum albumin over time would be prognostically more ominous than baseline values, and would be so even in a multicentre setting.
Methods We analysed the survival implications of baseline albumin and Δalbumin in a derivation cohort of 246 CHF outpatients (mean [±SD] age 68±12 years, LVEF 29±8%, 48% NYHA class >2) from University College London Hospital and then in a validation cohort of 148 CHF outpatients (age 69±12 years, LVEF 28±10%, 41% NYHA class >2) from Imperial Healthcare (St Marys Hospital and Hammersmith Hospital, London).
Results In the derivation cohort, 51 (21%) patients died over 13 months. Baseline albumin independently predicted mortality (HR 0.89, 95% CI 0.84 to 0.94, χ^2:18, p<0.0001). However, Δalbumin (unadjusted HR 0.89, 95% CI 0.84 to 0.92, χ^2:53, p<0.0001) was even more predictive (Difference in ROC AUC for baseline vs Δalbumin 0.16, p<0.001) and did so independently of all covariates including baseline albumin. A reduction in albumin > 6 g/l optimally predicted death (ROC AUC 0.82, p<0.0001) and conferred a sixfold escalated risk of mortality (HR 6.42, 95% CI 3.67 to 11.22, p<0.0001). In incremental prognostic analyses, the addition of Δalbumin to the strongest four variable model (baseline albumin, NYHA class, Δurea, Δhaemoglobin) dramatically augmented the χ^2 value (43 vs 84, p<0.0001). In the validation cohort, 43 (30%) patients died. Δalbumin (unadjusted HR 0.89, 95% CI 0.86 to 0.92, χ^2: 44, p<0.0001) was again prognostically superior to baseline albumin with a fall >6 g/l predicting an ~sixfold increased risk (HR 5.64, 95% CI 3.08 to 10.31, χ^2: 35, p<0.0001). Addition of Δalbumin to the strongest three variable model (baseline red cell distribution width, Δred cell distribution width, Δurea) also augmented the χ^2 value (51 vs 65, p<0.001).
Conclusions A fall in serum albumin over time consistently predicts an amplified risk of death in systolic CHF and enables simple and cheap risk stratification.

011 HBA1C AND MORTALITY IN DIABETIC INDIVIDUALS WITH HEART FAILURE: AN OBSERVATIONAL COHORT STUDY

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Background Controversy exists regarding the importance of glycaemic control in patients with type 2 diabetes mellitus (T2DM) and chronic heart failure (CHF) based on conflicting reports that had used a single baseline HbA1c.