The correlations of optimal AV delays by non-invasive (Finometer) systolic blood pressure (SBP) vs invasive measures were as follows; aortic SBP, \( r^2 = 0.96, p < 0.01 \); aortic flow velocity, \( r^2 = 0.81, p < 0.01 \); LV dP/dt\(_{\text{max}}\), \( r^2 = 0.68, p < 0.01 \).

**Conclusions** During acute biventricular pacing, at a fixed heart rate, changing the AV delay affects the cardiac mechaenoenergetics. When an AV delay improves external cardiac work, compared to LBBB or a physiologically too short AV delay (eg, AV 40 ms), it also increases the myocardial oxygen consumption. However, only 1% more energy is consumed per 1.6% more external work (pressure\( \times \)flow) done; as a result cardiac efficiency improves. Haemodynamic optimisation of AV delay can be achieved with high precision using non-invasive beat-to-beat pressure measurements. This should enable routine haemodynamic optimisation (easily automated) of CRT devices in clinical practice.

**89 ELECTROMECHANICAL INTERACTION IN PATIENTS UNDERGOING CARDIAC RESYNCHRONISATION THERAPY: COMPARISON OF INTRACARDIAC ACTIVATION MAPS AND EARLY SEPTAL CONTRACTION IN LEFT BUNDLE BRANCH BLOCK**

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**Introduction** Early inward motion and thickening/thinning of the ventricular septum associated with left bundle branch block (LBBB) is known as the septal flash (SF). Correction of SF corresponds with CRT response. We hypothesise that electromechanical interactions induced by SF are associated with functional changes in conductivity and a “U-shaped” activation pattern. Characterising the spatio-temporal relationship between electrical and mechanical events may explain why patients with a SF respond to CRT. Methods 15 patients (65±10 years, 10 men) with severe heart failure (EF 22.8±5.8%) undergoing CRT underwent echocardiography and non-contact mapping (NCM) pre-implant. Presence and extent of a SF was defined visually and with M-mode and fused with NCM bull’s eye plots of endocardial activation patterns. LV dP/dt\(_{\text{max}}\) was measured during different pacing modes.

**Results** Five patients had a large SF, four small SF and four no SF. Patients with large SF had areas of conduction block in non-infarcted regions whereas those with small or no SF did not (Abstract 89 figure 1). Patients with large SF had greater acute response to left ventricular (LV) and biventricular (BIV) pacing vs those with small/no SF (% increase dP/dt 28±14% vs 11±19% for LV pacing and 42±28% vs 22±21% for BIV pacing). The lines of conduction block disappeared after LV and BIV pacing, while remaining present with RV pacing (Abstract 89 figure 2). Abstract 89 figure 1 Patient with a large SF. Unipolar isochronal map with NCM electrograms showing fragmented signals (development of split potentials) indicating a reduction of conduction and inability to cross throughout the inferior region. The NCM mapping electrograms show the criteria used by Auricchio et al to define block, with the emergence of R-wave, smallest and earliest at the superior part of the block (where area of block begins) with largest negative peak. Bold white arrows on the electrogram indicate how the electrical activation spreads superficially in a U-shape pattern leading to the development of split potentials. Abstract 89 figure 2 Activation maps of patient with a large SF. Row A, baseline with area of block and late anterior breakthrough. Row B, RV pacing showing the area of anterior block remains. Row C, BIV pacing. Functional conduction block has disappeared.

**90 INVASIVE ACUTE HAEMODYNAMIC RESPONSE TO GUIDE LV LEAD IMPLANTATION PREDICTS CHRONIC REMODELLING IN PATIENTS UNDERGOING CARDIAC RESYNCHRONISATION THERAPY**

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**Introduction** Cardiac resynchronisation therapy (CRT) reduces mortality and morbidity in heart failure patients, however up to 30% of patients do not derive symptomatic benefit. Higher proportions do not remodel. Multi-centre trials have shown echocardiographic techniques are poor at improving response rates. We hypothesised that the degree of acute haemodynamic response (AHR) at implant can predict which patients remodel. We evaluated the relationship between AHR and reverse remodelling (RR) in CRT. Methods 35 patients undergoing CRT (21 dilated & 12 ischaemic cardiomyopathy) were studied. Left ventricular (LV) volumes were assessed pre and post CRT. AHR (LV-dP/dt\(_{\text{max}}\)) was assessed at implant using a pressure wire in the LV cavity. The LV lead was placed in potential target veins and the largest percentage rise in LV-dP/dt\(_{\text{max}}\) from baseline (AAR or RV pacing with atrial fibrillation) to DDDLV was used to determine optimal LV lead position. RR was defined as reduction in LV end systolic volume (ESV) ≥15% at 6 months.

**Results** LV-dP/dt\(_{\text{max}}\) increased significantly from baseline (801±194 mm Hg/s to 924±205 mm Hg/s (p<0.001)) with DDDLV pacing for the optimal LV lead position. There was a significant difference in the percentage rise in LV-dP/dt\(_{\text{max}}\) between the best and worst LV lead position (Abstract 90 figure 1). LV ESV...
However, a proportion of patients do not derive benefit from established treatment for patients with advanced heart failure (HF). Background Cardiac resynchronisation therapy (CRT) is an established treatment for patients with advanced heart failure (HF). However, a proportion of patients do not derive benefit post implantation of CRT. Despite an established predictive role in HF, the significance of RV dysfunction in gauging clinical benefit from CRT has not been investigated. Cardiovascular magnetic resonance (CMR) is an important tool in the assessment of HF and is considered the gold-standard in estimating RV function. We used this technique to assess the impact of RV dysfunction on clinical outcomes following CRT implantation.

Methods We evaluated 48 consecutive patients attending a heart failure pacing clinic who had a CMR study within 6 months prior to CRT implantation. Clinical, biochemical, ECG and imaging data were collected. Biventricular function and myocardial scar were assessed by CMR including gadolinium enhancement. The primary end-point was a composite of all cause mortality (ACM) or unplanned cardiovascular hospitalisation.

Results The mean age was 64.5±12.7 years. HF was ischaemic in 42% of patients, and 85% were in NYHA class III/IV at the time of implantation. Atrial fibrillation/flutter was found in 27% of patients. The mean LVEF estimated by CMR was 27±5%, while the median RV EF was 52% (IQR 38%–65%). The mean tricuspid annular plane systolic excursion (TAPSE) was 14.0±6.0 mm, and the mean pulmonary artery pressure (on echocardiography) was 57±10 mm Hg. Ten patients (21%) met the primary end-point over a mean follow-up of 28.6 months. On time-to-event analysis, only atrial fibrillation (HR 4.3, p=0.02) and RV dysfunction, ie, reduced RV EF (HR 0.96 per 1% EF, p=0.01) or TAPSE (HR 0.80 per mm, p<0.01) were independent predictors of the primary end-point. Atrial fibrillation and low RV EF were the only independent predictors of mortality (p=0.03 and 0.04, respectively).

Conclusions RV dysfunction is an independent predictor of adverse clinical outcomes following CRT. The assessment of RV function may be considered in patient selection for CRT implantation.

Conclusions Acute haemodynamic response to LV pacing is useful for predicting which patients are likely to remodel in response to CRT both for DCM and ICM. There is much variation in the rise in LV-dP/dtmax depending on LV lead position. Using acute haemodynamic response measured with a pressure wire during CRT implantation has the potential to guide LV lead positioning and improve response rates in the future.