

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/dtmax,  $r^2=0.68$ ,  $p<0.01$ .

**Conclusions** During acute biventricular pacing, at a fixed heart rate, changing the AV delay affects the cardiac mechanoenergetics. 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.

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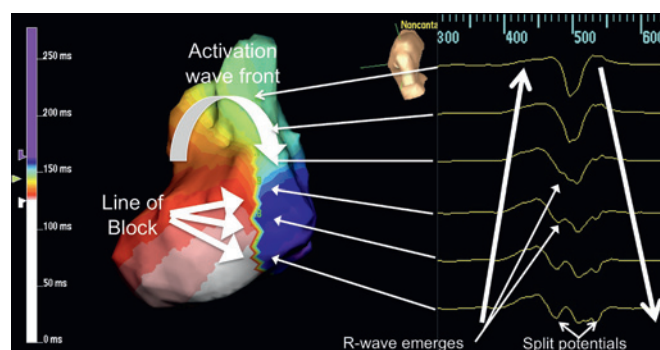
# **ELECTROMECHANICAL INTERACTION IN PATIENTS UNDERGOING CARDIAC RESYNCHRONISATION THERAPY: COMPARISON OF INTRACARDIAC ACTIVATION MAPS AND EARLY SEPTAL CONTRACTION IN LEFT BUNDLE BRANCH BLOCK**

doi:10.1136/heartjnl-2011-300198.89

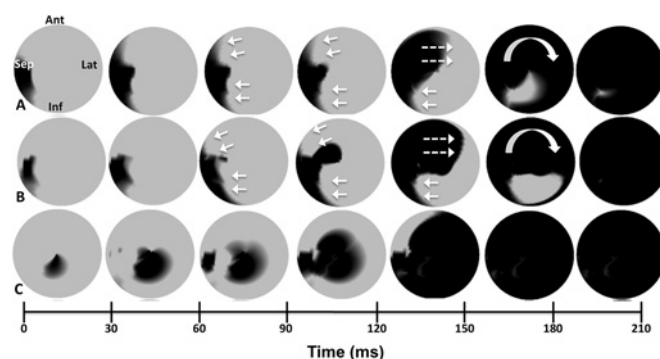
<sup>1</sup>S G Duckett, <sup>2</sup>O Camara, <sup>1</sup>M Ginks, <sup>3</sup>J Bostock, <sup>1</sup>P Chinchapatnam, <sup>1</sup>M Serresant, <sup>2</sup>A Pashaei, <sup>3</sup>J S Gill, <sup>3</sup>G Carr-White, <sup>2</sup>A F Frangi, <sup>1</sup>R S Razavi, <sup>2</sup>B H Bijnens, <sup>3</sup>C A Rinaldi. <sup>1</sup>Kings College London, London, UK; <sup>2</sup>UPF, Barcelona, Spain; <sup>3</sup>Guy's and St Thomas' Hospital, London, UK

**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 13 patients (63 $\pm$ 10 years, 10 men) with severe heart failure (EF 22.8 $\pm$ 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/dtmax 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 $\pm$ 14% vs 11 $\pm$ 19% for LV pacing and 42 $\pm$ 28% vs 22 $\pm$ 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 superiorly 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.



Abstract 89 Figure 1



Abstract 89 Figure 2

**Conclusion** A strong interaction exists between electrical activation and mechanical deformation of the septum. Correction of both mechanical synchrony and the functional conduction block by CRT may explain the large positive response in patients with a SF.

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

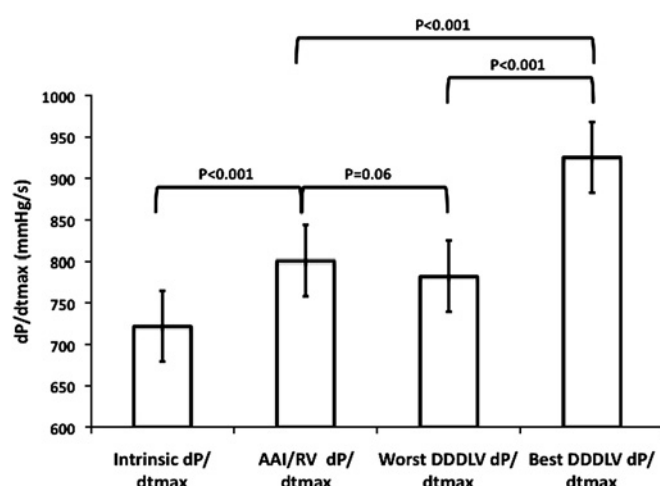
doi:10.1136/heartjnl-2011-300198.90

<sup>1</sup>S G Duckett, <sup>1</sup>M Ginks, <sup>1</sup>A Shetty, <sup>2</sup>J Bostock, <sup>2</sup>J S Gill, <sup>2</sup>S G Hamid, <sup>2</sup>S Kapetanakis, <sup>2</sup>E Cunliffe, <sup>1</sup>R S Razavi, <sup>2</sup>G Carr-White, <sup>2</sup>C A Rinaldi. <sup>1</sup>Kings College London, London, UK; <sup>2</sup>Guy's and St Thomas' Hospital, London, UK

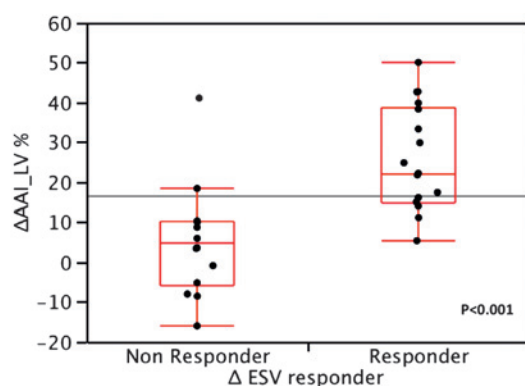
**Introduction** Cardiac resynchronisation therapy (CRT) reduces mortality and morbidity in heart failure patients, however up to 30% of patients do not derive symptomatic benefit. Higher proportions do not remodel. Multi-centre trials have shown echocardiographic techniques are poor at improving response 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 33 patients undergoing CRT (21 dilated & 12 ischaemic cardiomyopathy) were studied. Left ventricular (LV) volumes were assessed pre and post CRT. AHR (LV-dP/dtmax) 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/dtmax from baseline (AAI or RV pacing with atrial fibrillation) to DDDLIV was used to determine optimal LV lead position. RR was defined as reduction in LV end systolic volume (ESV)  $\geq$ 15% at 6 months.

**Results** LV-dP/dtmax increased significantly from baseline (801 $\pm$ 194 mm Hg/s to 924 $\pm$ 203 mm Hg/s ( $p<0.001$ )) with DDDLIV pacing for the optimal LV lead position. There was a significant difference in the percentage rise in LV-dP/dtmax between the best and worst LV lead position (Abstract 90 figure 1). LVESV

decreased from  $186 \pm 68$  ml to  $157 \pm 68$  ml ( $p < 0.001$ ). 18 (56%) patients exhibited RR. There was a significant relationship between percentage rise in LV-dP/dtmax and RR for DDDLIV pacing ( $p < 0.001$ ) (Abstract 90 figure 2). A similar relationship for AHR and RR in DCM and ICM ( $p = 0.01$  &  $p = 0.006$ ) was seen.



Abstract 90 Figure 1



Abstract 90 Figure 2

**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 implant has the potential to guide LV lead positioning and improve response rates in the future.

## 91 RIGHT VENTRICULAR DYSFUNCTION IDENTIFIES CLINICAL OUTCOMES FOLLOWING CARDIAC RESYNCHRONISATION THERAPY

doi:10.1136/heartjnl-2011-300198.91

<sup>1</sup>K Guha, <sup>2</sup>F Alpendurada, <sup>2</sup>S Prasad, <sup>2</sup>T McDonagh, <sup>1</sup>M R Cowie, <sup>2</sup>R Sharma. <sup>1</sup>Royal Brompton Hospital, Imperial College, London, UK; <sup>2</sup>Royal Brompton Hospital, London, UK

**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  $64.5 \pm 12.7$  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  $27 \pm 8\%$ , while the median RVEF was 52% (IQR 35%–63%). The mean tricuspid annular plane systolic excursion (TAPSE) was  $14.0 \pm 6.0$  mm, and the mean pulmonary artery pressure (on echocardiography) was  $37 \pm 10$  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 4.8,  $p = 0.02$ ) and RV dysfunction, ie, reduced RVEF (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 RVEF 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.

## 92 IDENTIFYING PATIENTS WITH CHRONIC HEART FAILURE: A COMPARISON OF THE GOLD STANDARDS FRAMEWORK WITH A CLINICAL PROGNOSTIC MODEL

doi:10.1136/heartjnl-2011-300198.92

<sup>1</sup>K K Haga, <sup>2</sup>S A Murray, <sup>3</sup>J Reid, <sup>3</sup>A Ness, <sup>3</sup>M O'Donnell, <sup>4</sup>D Yellowlees, <sup>5</sup>M A Denver. <sup>1</sup>University of Edinburgh, School of Medicine and Veterinary Medicine, Edinburgh, UK; <sup>2</sup>Primary Palliative Care Research Group, University of Edinburgh, Edinburgh, UK; <sup>3</sup>Heart Failure Nursing Service, NHS Lothian, Edinburgh, UK; <sup>4</sup>University of Edinburgh, Edinburgh, UK; <sup>5</sup>Department of Cardiology, University of Edinburgh, School of Medicine and Veterinary Medicine, Edinburgh, UK

**Introduction** Heart failure has a worse survival rate than many common cancers, yet few patients receive any palliative care input during the course of their illness. One of the main difficulties in providing palliative care for heart failure patients is the uncertainty around the course of the disease and the patient's life expectancy. The aim of this study was to compare the "Gold Standards Framework" (GSF) criteria, which were developed to determine the need for palliative care in non-cancer patients, with the "Seattle Heart Failure (SHF) Model", which provides a method of calculating a patient's predicted mean life expectancy using physiological variables.

**Methods** Chronic heart failure patients, in NYHA class III or IV, who were being managed in the specialist, heart failure nursing service, were identified from a clinical heart failure database. GSF criteria were assessed by interviewing the specialist nurse responsible for each patient's care. Clinical data required for the SHF model were obtained from two, online databases and were used to estimate mean life expectancy and predicted mortality at 1 year. Patients were then followed up, at 1 year, to evaluate; 1) all cause mortality, 2) place of death, and 3) the sensitivity and specificity of the GSF and SHF to predict death at 1 year.

**Results** 138 NYHA III-IV patients were identified from a total of 368 patients currently managed within the specialist nurse service; 66% were male, and the mean age was 77 years. GSF criteria,