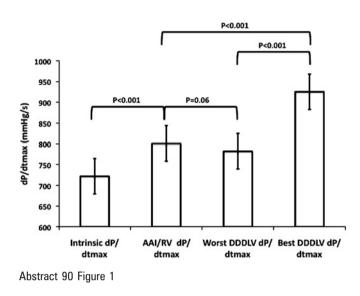
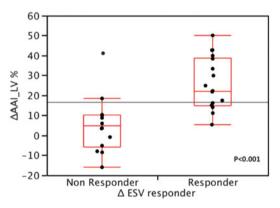
decreased from  $186\pm68$  ml to  $157\pm68$  ml (p<0.001). 18 (56%) patients exhibited RR. There was a significant relationship between percentage rise in LV-dP/dtmax and RR for DDDLV pacing (p<0.001) (Abstract 90 figure 2). A similar relationship for AHR and RR in DCM and ICM (p=0.01 & p=0.006) was seen.





Abstract 90 Figure 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 haemo-dynamic 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

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**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  $64.5\pm12.7$  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  $27\pm8\%$ , while the median RVEF was 52% (IQR 35%-63%). The mean tricuspid annular plane systolic excursion (TAPSE) was  $14.0\pm6.0$  mm, and the mean pulmonary artery pressure (on echocardiography) was  $37\pm10$  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

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**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,

identified 119/138 (86%) patients that met the minimum requirement for palliative care input. However, the SHF model predicted that only 6/138 patients (4.3%) had a predicted life expectancy of less than 1 year. Patients who met GSF criteria for palliative care had significantly more hospital admissions (p=0.001) and had significantly lower predicted survival rates at 1 year (p=0.038) than those patients that did not meet GSF criteria. At follow-up, 43/138 patients had died (31%). Of these, 58% (25/43) died in hospital, following an acute admission. The sensitivity and specificity for the GSF and SHF model were 22%/83% and 98%/12% respectively. Overall, the patients renal function (eGFR<35 ml/min) was the best predictor of mortality, (sensitivity/specificity=82%/56%).

**Discussion** Neither the GSF nor the SHF were very accurate in predicting which patients were in the last year of life, in this selected sample. Despite the increasing drive towards palliation in heart failure, clinicians are still faced with a substantial prognostic barrier. Therefore, the progress of palliative care in heart failure patients may require a shift away from the traditional "end of life" model developed in cancer treatment, and focus instead on a patient's increasing needs coupled with an understanding that death, itself, may remain unpredictable.

## 93 OPTIMAL MEDICAL THERAPY IN HEART FAILURE: IS THERE SPACE FOR ADDITIONAL HEART RATE CONTROL?

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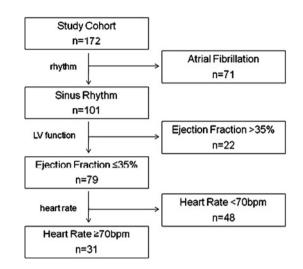
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Introduction Current evidence suggests that heart rate (HR) may serve both as a modifiable risk factor, and as a disease modifying variable in patients with impaired left ventricular (LV) systolic function. The systolic heart failure (HF) treatment with  $I_{\rm f}$  inhibitor ivabradine trial (SHIFT) for example recently demonstrated significantly improved outcomes in otherwise optimally treated HF patients following additional HR reduction with ivabradine. We therefore estimated the number of patients who after optimisation of conventional HF medications may be suitable for additional HR reduction.

**Methods** We performed a retrospective analysis from two HF clinics where patients are referred for nurse lead, protocol-guided optimisation of conventional HF therapies. Data on patient demographics and classification of HF including; severity (ejection fraction>35% vs ejection fraction $\leq$ 35%), functional limitation (New York Heart Association; NYHA class), and cause (ischaemic vs non-ischaemic) were recorded. In addition, we collected data on patient's resting pulse (absolute value and rhythm: sinus vs atrial fibrillation), and blood pressure at the first and last clinic visits. Between the two clinic visits, patients underwent protocol-guided forced up-titration of standard neurohormonal HF therapies. We also collected data on the maximal tolerated doses of beta blocker ( $\beta$ B), ACE inhibitor (ACE-I) or angiotensin receptor blocker (ARB), and the reasons for the inability to achieve target doses of  $\beta$ B.

**Results** Of 172 consecutive patients referred for optimisation of HF therapies (age 71±13 yrs, 67% male), 71 (41%) were in atrial fibrillation. Of the patients in sinus rhythm, 78% had severe LV systolic dysfunction (Abstract 93 figure 1). Overall, 145 of 172 patients (83%) tolerated  $\beta$ B therapy; of these, 39% achieved the target maximal dose, 57% at least half target dose, and 4% less than half of the target dose of  $\beta$ B. Reasons for failure to initiate  $\beta$ B (n=27, 17%) included; severe and limiting hypotension (48%), intractable lethargy (26%), and hospitalisation with worsening airways disease (26%). ACE-I/ARB, aldosterone antagonists, and digoxin were

tolerated in 92%, 30%, and 18% of patients respectively (Abstract 93 table 1). Resting heart rate and blood pressure before and after optimisation of medical therapy are shown in Abstract 93 table 2.



Abstract 93 Figure 1 Heart Failure Patients Potentially Suitable for Additional Heart Rate Reduction After Optimisation of Standard Medical Therapy.

Abstract 93 Table 1 Patient Characteristics (n=172)

NYHA Class (%)	
1	10.5
II	62.2
III	26.2
IV	1.1
HF aetiology (%)	
Ischaemic	57
Non-ischaemic	43
LV function (%)	
Ejection Fraction $\leq$ 35%	92.4
Ejection Fraction >35%	7.6
Cardiac rhythm (%)	
Sinus	58.7
Atrial Fibrillation	41.3
Medication use (%)	
β-blockers	83
ACE-I/ARBs	92
Aldosterone antagonists	30
Digoxin	18

Abstract 93 Table 2 Haemodynamic profiles before and after optimisation of medication

	First Clinic Visit (pre-optimisation)	Final Clinic Visit (post-optimisation)	p value
Resting Heart Rate (beats/min)	73.8±14.8	67.3±9.5	< 0.001
Systolic Blood Pressure (mm Hg)	120±19.6	115.1±18.0	< 0.001
Diastolic Blood Pressure (mm Hg)	71.7±11.6	67.2±10.4	< 0.001

**Conclusions** Of 172 unselected patients attending HF clinics for optimisation of medical therapy, ~50% are in sinus rhythm with an ejection fraction  $\leq$ 35%. Despite forced optimisation of medical therapy, half of these patients have a resting heart rate  $\geq$ 70 beats/minute. Overall, ~1 in every 3 patients attending a heart failure clinic may be suitable for additional heart rate control.