clinical impact on morbidity and mortality of patients with chronic HF (CHF) remains to be determined.

Purpose To evaluate the prevalence, clinical associations and prognostic value of three different malnutrition indices in a large cohort of patients referred to a community HF clinic with suspected HF.

Methods Consecutive referrals were analysed. HF was defined as signs or symptoms and evidence of cardiac dysfunction, either a reduced left ventricular ejection fraction at echocardiography (LVEF, <50%) or raised NTproBNP (>125 ng/L). Patients were screened for malnutrition using three common malnutrition indices: the geriatric nutritional risk index (GNRI), the controlling nutritional status (CONUT) score and the prognostic nutritional index (PNI).

Results Of the 5012 patients who attended between 2000 and 2016, HF was confirmed in 3386 (61% males, median age 75 (interquartile range (IQR): 67–81) years, median NTproBNP 1573 (IQR: 702–2799) ng/L). Of these, 1198 (35%) and 1458 (43%) patients had HF with reduced (HeFREF, LVEF<40%) and normal (HeFNEF, LVEF 50%) ejection fraction, respectively. According to the GNRI (91%), CONUT score (>4) and PNI (38%), 6.7%, 10.0% and 7.5% patients were moderately to severely malnourished, respectively.

Compared to those with normal nutritional status, malnourished patients were older, had lower body mass index (BMI), worse symptoms and renal function; they were also more likely to have atrial fibrillation, anaemia and reduced mobility.

During a median follow-up of 1573 days (interquartile range: 702–2799 days), 1723 (50.9%) patients died. In multivariable models, indices of malnutrition were powerful predictors of mortality. In a multivariable model that included all the malnutrition indices, only GNRI was independently associated with increased risk of mortality (Hazard ratio (95% confidence interval): 1.25 (1.12–1.39), $p<0.001$ for worsening malnutrition category of GNRI).

Conclusion Moderate or severe malnutrition is highly prevalent amongst patients with CHF and is strongly related to mortality.

7 IMPLANTABLE CARDIAC DEVICE GENERATOR CHANGE PATHWAYS: AN OPPORTUNITY FOR OPTIMISING DEVICE PRESCRIPTION

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Introduction Implantation of cardiac devices including permanent pacemakers (PPM), cardiac resynchronisation therapy with defibrillators (CRT-D) or without (CRT-P) is increasingly common. Follow up, including arranging generator change, is frequently carried out by cardiac physiologists, with minimal physician input. Many patients clinical status will change during the lifetime of the device. Generator change is an opportunity to review the original device prescription. We report the impact of introducing routine screening prior to generator change into an established comprehensive device programme.

Methods Two pathways were implemented. Patients with a PPM due generator change were screened by cardiac physiologists using a proforma assessing symptoms, QRS duration and right ventricular (RV) pacing burden. Those with heart failure symptoms, in particular breathlessness, and a prolonged native

QRS or >40% RV pacing had an echocardiogram and multi-disciplinary team (MDT) discussion within 2 weeks. If device upgrade was potentially indicated the patient was reviewed in clinic prior to generator change.

The goal was to discuss all patients with an ICD/CRT-D due generator change at MDT. Those with changes in clinical status that may affect the appropriateness of ongoing defibrillator therapy were reviewed in clinic.

Results The first 11 months, October 2015 to August 2016, following implementation of these pathways were studied. 189 consecutive patients (145 PPM, 20 CRT-P and 24 CRT-D/ICD), mean age of 77, were included.

The pathways resulted in a change in the type of device therapy in 8 patients; 6/145 (4%) of those due PPM and 2/24 (8%) due CRT-D/ICD generator change (table 1).

145 (100%) of PPM patients were proforma screened. 139/145 PPM patients (96%) went on to have PPM generator change. 17 (12%), identified as potential candidates for upgrade to CRT, had echocardiography. 5 (3%) of these had severe LV impairment and underwent physician and MDT review. 4 patients (3%) were ultimately upgraded to CRT devices. CRT was not appropriate in 1 patient. In 2 (1%) patients ongoing device therapy of any kind was felt to be inappropriate (1 potential upgrade and 1 PPM box change) and no procedure was undertaken.

10/24 (42%) patients due a CRT-D/ICD generator change were reviewed at MDT. Of these 2 (20% of those reviewed) went on to have their devices downgraded from CRT-D to CRT-P after informed discussion.

Conclusions Physiologist-led device follow up can be effectively extended to include robust screening of patients to ensure they get the most appropriate device at the time of generator change. Excellent rates of PPM screening were achieved whereas ICD screening needs to be improved.

Relatively few patients had their destination device changed as a result of these pathways. However there is significant morbidity associated with receiving inappropriate device therapy, justifying implementation of a simple screening process to improve care.

Abstract 7 Table 1 Outcomes for patients due implantable cardiac device generator change after the implementation of routine screening

Outcomes	Number of patients
Type of device unchanged	181
Upgrade from PPM to CRT-P	2
Upgrade from PPM to CRT-D	2
Downgrade from CRT-D to CRT-P	2
Device therapy withdrawn	2

8 THE HF-CGM STUDY: AN ANALYSIS OF CARDIOGONIOMETRIC AXES IN PATIENTSWITH CARDIAC RESYNCHRONISATION THERAPY

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Introduction The HF-CGM study is a proof-of-principle study to investigate whether cardiogoniometry (CGM), a three-

dimensional electrocardiographic method, can differentiate between pacing modes in patients with cardiac resynchronisation therapy (CRT).

Methods At a tertiary cardiology centre, CGM recordings were performed using four pacing modes: no pacing; right ventricular (RV) pacing; left ventricular (LV) pacing and biventricular (BIV) pacing. Three orthogonal CGM planes orientated to the long axis (XY), the frontal plane (YZ) and the short axis (XZ) of the heart were constructed (see figure), and the direction of the QRS axis was calculated for each pacing mode in each plane. During BIV pacing, the direction of CGM QRS axis was compared between patients with optimal and non-optimal 12-lead ECG pacing variables. Optimally paced 12-lead ECG variables were defined as an R/S ratio greater than or equal to 1 in V₁ and/or R/S ratio less than or equal to 1 in lead I.

Results Eleven participants (aged 77.4 ± 11.5 ; 63.6% male, LVEF $31 \pm 6\%$) were consecutively recruited. Only QRS axis measured in the XY plane could distinguish between LV and BIV pacing vs. no pacing ($p=0.005$ and $p=0.001$ respectively). Mean QRS axis and 95% confidence intervals (CI) for each pacing mode is shown in the table. Mean QRS axis in the XY plane with pacing off and during RV pacing was leftwards and basal; LV pacing was apical; and BIV pacing was rightwards and basal. There was a marked difference in the direction of QRS axis between patients with optimal vs. non-optimal paced QRS morphology in the XY plane (rightwards and basal vs inconsistent).

Abstract 8 Table 1

	XY plane	YZ plane	XZ plane
No	-23	147	-8
pacing	(95% CI: -35 to 11)	(95% CI: 117 to 177)	(95% CI: -45 to 29)
RV	-52	144	-10
pacing	(95% CI: -66 to -40)	(95% CI: 136 to 152)	(95% CI: -23 to 4)
LV	148	-24	74
pacing	(95% CI: 88 to -152)	(95% CI: -166 to 113)	(95% CI: 7 to 157)
BIV pacing	-130	150 (95% CI: 29 to -88)	-91
	(95% CI: 161 to -62)		(95% CI: 102 to 71)

Abstract 8 Figure 1 Cardiac CT sections taken in the same approximate sections as the CGM planes – XY plane (panel A), YZ plane (panel B), XZ plane (panel C). Green shading represents the right ventricle; yellow shading represents the left ventricle.

Conclusions CGM recorded in the XY plane can accurately detect differences between ventricular pacing sites. Further studies are needed to determine whether CGM has clinical utility in optimising patients undergoing CRT.

9

IDENTIFICATION OF A POTENTIAL CAUSE OF NON-RESPONSE TO BIVENTRICULAR PACING USING INTRAVENOUS ADENOSINE

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Aim We aim to evaluate the efficacy and safety of determining the presence of true biventricular pacing by administration of intravenous adenosine. This aims to determine whether non-response to biventricular pacing therapy is partially explained by over-counting of the percentage of pacing therapy by the presence of fusion beats. We compared the proportion of patients with fusion beats in non-responders compared to responders

Methods This will be a single centre, prospective, cohort study. 71 consecutive patients with implanted cardiac resynchronisation devices between July 2006 and November 2015 were identified. Patients underwent the adenosine trial at least 12 months post CRT device implantation to detect the presence of potentially ineffective biventricular pacing. This was compared with the recorded percentage of biventricular pacing reported during device interrogation. Correlation was made between response to CRT therapy and presence of effective biventricular pacing.

Results Preliminary results from 24 patients demonstrate that symptomatic adenosine administration was successful in identifying true effective biventricular paced beats. This has allowed us to identify patients with fusion beats. 8 patients were non-responders to CRT, whereas 16 patients were responders. 50% ($n=4$) of non-responders and 12.5% ($n=2$) of responders had definite electrocardiography (ECG) changes through the symptomatic adenosine administration, showing that a percentage of the paced beats counted by the CRT device may in fact be fusion beats. Figure 1 shows the clear change in the morphology of the QRS complex in one of the non-responders.

Figure 1 Electrocardiogram of a non-responder during symptomatic adenosine administration

Conclusion Preliminary results suggest that symptomatic adenosine administration is successful in identifying true effective biventricular paced beats which may be overestimated by CRT device interrogation. Correlation with response to CRT is yet to be established, but in 50% of the non-responders we identified a definite change in the morphology of the paced rhythm during the symptomatic response to adenosine. This raises a question of whether the poor response to CRT for these patients is actually due to the insufficient percentage of effective biventricular pacing ($<98\%$).