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ELECTROPHYSIOLOGY

Magnetic resonance criteria for future trials of cardiac resynchronization therapy

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Current patient selection criteria for Cardiac Resynchronization Therapy (CRT), an efficacious treatment for heart failure, include no measure of disconjugate cardiac contractility other than prolonged QRS on electrocardiogram. Using cardiac magnetic resonance imaging, we examined the roles of cardiac asymmetry, asynchrony, and circumferential strain in DCC with the principal aim of generating a robust numerical index for use in future trials of CRT. Standard cardiac magnetic resonance imaging was done on a GE 1.5 Tesla Signa LX MRI clinical scanner (GE Healthcare, Milwaukee, WI, USA) and analyzed by MASS Analysis (MEDIS, Leiden, The Netherlands). The methods were evaluated in eleven patients with advanced heart failure due to ischemic and non-ischemic cardiomyopathy, who did not qualify under current criteria for CRT, five CRT candidates pre-op and eleven normal subjects. Using *t*-test and standardized differences (SD = sd/diff, Power (N) = number of patients to reach $p < .05$) we determined efficacy. Indices of asymmetry and asynchrony (I_{sm} and I_{sn}, respectively) could be measured with accuracy and provided excellent statistical power when used as surrogate markers to delineate heart failure and CRT patients from control subjects. Asymmetry and asynchrony in heart contraction are both critical components of dilated cardiomyopathy that can be improved by CRT. Magnetic resonance asynchrony is efficacious in screening patients and should now be compared with recently published echocardiography data to improve outcome for this costly but valuable therapy.

Key Words: Heart failure; Magnetic resonance imaging; Myocardial contraction; Cardiac resynchronization therapy; Ejection fraction; Efficacy

1. Introduction

Approximately 400,000 new cases of congestive heart failure are diagnosed per year in the United States (1). CRT is a recent interventional method of treatment for patients with congestive heart failure (2, 3). Also known as bi-ventricular or multi-site pacing, CRT involves the installation of pacing leads to both the left and right sides of the heart to synchronize myocardial contraction. As heart failure progresses and becomes more refractory to medical treatment, CRT may prove a very attractive first-line therapy for heart failure patients who might otherwise require transplantation.

Recent studies have revealed that CRT reduces mortality by heart failure by 30 to 50 percent (4, 5) and can lower risk of hospitalization by up to 29 percent (5). Despite these benefits, a significant portion of CRT recipients do not respond well to the therapy. It was estimated in the 2002 Multicenter Insync Randomized Clinical Evaluation (MIRACLE) (6) that only 68 percent of the patients participating in the trial responded positively to the procedure.

This lack of response in patients may be the result of imprecise qualification criteria for CRT. The current criteria for CRT candidacy are classification of New York Heart Association (NYHA) Class III or IV heart failure, a diagnosis of advanced heart failure HF due to ischemic or non-ischemic cardiomyopathy, an EF less than 35 percent, (normal EF = 50–70%), an ECG QRS complex of over 130 milliseconds (normal $\leq$ 100 ms), and a LVEDD > 55 mm (7, 8).

Despite occasional suggestions in the literature (4), abnormal ventricular wall motion has hitherto not been a criterion for CRT candidacy. Though ejection fraction is a global measure of cardiac contractility, it is an indirect measure of left ventricular contraction and does not rely on measurements of asynchrony and asymmetry, which are direct measures of LV wall motion. CMR imaging is superior to echocardiography in depiction of wall motion and contrast resolution (12, 13) and has already shown promise in wall motion analysis of myocardial strain measurement employing MR tagging and spatial modulation of magnetization (SPAMM) (13–15). The aim of this study was to use the

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Table 1A. Study population demography: CMR values

CMR measures	Control (n = 11)	HF (n = 11)	CRT (n = 5)	p values (HF vs. CRT)
Mean age	54.1 ± 11.3	61.6 ± 15.1	70.4 ± 9.3	NS
Age range	35–76	37–92	60–83	NS
Male/female ratio	6/5	9/1	3/4	NS
Ejection fraction (%)	69.2 ± 6.8	23.7 ± 8.9	24.4 ± 6.3	.89
Stroke volume (ml)	60.4 ± 15.5	43.8 ± 9.7	45.7 ± 13.7	.84
End diastolic volume (ml)	88.1 ± 23.7	219.7 ± 110.3	190.6 ± 44.4	.69
End systolic volume (ml)	27.7 ± 11.3	175.8 ± 113.1	144.7 ± 39.4	.74

This table shows the demography of the study population, group mean values and standard deviations.
NS = not significant.

extensive capabilities of CMR to develop a method of quantifying asymmetry and asynchrony in left ventricular myocardial contraction, with the goal of improving criteria for CRT qualification.

Indices of DCC were quantified in terms of I_{sm}, I_{sn} and Cs. The goals of this study were to determine normal CMR ranges of DCC, to test whether values of DCC as calculated by CMR can distinguish between control subjects and HF patients, and to quantify measures of DCC in a group of patients who qualify for CRT. During the preparation of this report, echocardiography and tissue Doppler imaging have demonstrated the capability of measuring values of intra-ventricular and inter-ventricular asynchrony (9–11); however, a formal index by which irregular myocardial contraction or DCC can be measured has not been developed.

2. Materials and methods

2.1. *Study population*

Twenty-seven subjects were examined by CMR (Table 1A). Measures of I_{sm} and I_{sn} in left ventricular heart contraction were developed in 11 normal subjects (6 males, 5 females, mean age 51.5 ± 11.0, range 35–76 years). These indices of I_{sm} and I_{sn} (criterion: $\geq$ mean + 2 SD) were then applied to 11 patients clinically diagnosed with heart failure (9 males, 2 female, mean age 66 ± 15, age range 37–92). Finally, the same measurements were applied to 5 patients with HF (2 males, 3 females, mean age 67 ± 9, age range 60–81) who were also qualified candidates for CRT, meeting the 2001 and 2002 ACC/AHA/NASPE criteria for CRT [EF < 35%, QRS>120 ms (2001) (3), QRS > 130 ms (2002), LVEDD > 55

Table 1B. Patient demographics

Age	Sex	Cardiomyopathy	EF from ECHO (Pre-op)	LVEDD (mm) from ECHO (Pre-op)	QRS Duration (Pre-op)	NYHA Class (Pre-op)	EF from MRI (Pre-op)
CRT patients							
60	M	Ischemic	20–25	63	133	III/IV	20
69	F	Non-ischemic	20	63	154	III	26
60	F	Non-ischemic	20	68	140	III	30
64	M	Ischemic	30	56	170	III	13
81	F	Ischemic	25–30	66	154	III	30
HF patients							
58	M	Non-ischemic	25–30	74	94	III/IV	25
61	M	Non-ischemic	20		146	III	7
77	F	Ischemic	26		110		26
64	F	Non-ischemic	30	72	108		26
92	M	Ischemic	30–34	71	154		33
70	M	Ischemic	30	65	141		25
65	M	Non-ischemic	45	85		I	35
49	M	Non-ischemic	25	75	83		24
37	M	Non-ischemic	10–15	68	98		13
72	M	Non-ischemic	25	83	154		26
79	M	Ischemic	35	64	176	I	26

This table demonstrates the HF and CRT patient populations and their individual NYHA class, QRS, Ejection Fraction, LVEDD (both Echo and MRI) data, indicating their ACC/AHA/NASPE qualifying criteria for cardiac resynchronization therapy.

mm (7, 8)]. CRT patient profiles are listed in Table 1B. Reproducibility of this study indicate that inter-observer error was ± 5 percent and repeated exam error was + 10 percent. All studies were successfully completed and approved by the Internal Review Board of Huntington Memorial Hospital and performed with patient consent.

2.2. *CMR image acquisition*

Studies were performed on a 1.5 Tesla Signa LX MRI clinical scanner (GE Healthcare, Milwaukee, Wisconsin). Exams were gated by a 4-lead ECG. Patients were scanned in the supine position (entering the MR scanner feet first) using a 4 element phased array cardiac coil while performing 10–15 second breath holds at end-expiration to minimize respiratory artifacts. Sagittal and long axis localizers were acquired to determine the orientation of the heart, after which 14–15 short axis slices covering the entire left ventricle from apex to base were acquired using steady state free precession (FIESTA) and gradient echo sequences (GRE). Tagged images of the heart were acquired in the short axis plane using a T2 weighted fast spoiled grass (FSPGR) sequence.

2.3. *Data analysis*

Images were transferred to an Advantage Windows (AW) workstation (GE Medical Systems). FIESTA or FSPGR short axis slices of the LV were analyzed by MR Analytical Software Systems (MASS) Analysis Plus (MEDIS Inc., Netherlands) (16) for determination of cardiac output, EF, SV, EDV, and ESV. Tagged data was analyzed using Harmonic Phase Magnetic Resonance Imaging (HARP) (Diagnosoft Inc., Maryland) (17) for Cs.

2.3.1. *Determination of DCC by asymmetry and asynchrony*

A contouring tool, Mass Analysis Plus, was used to delineate epicardial and endocardial surfaces and calculate myocardial wall thickness over the full cardiac cycle for two-dimensional slices of the LV from the base to the apex of the heart. Slices of the LV were divided into 6 equal segments numbered 1–6

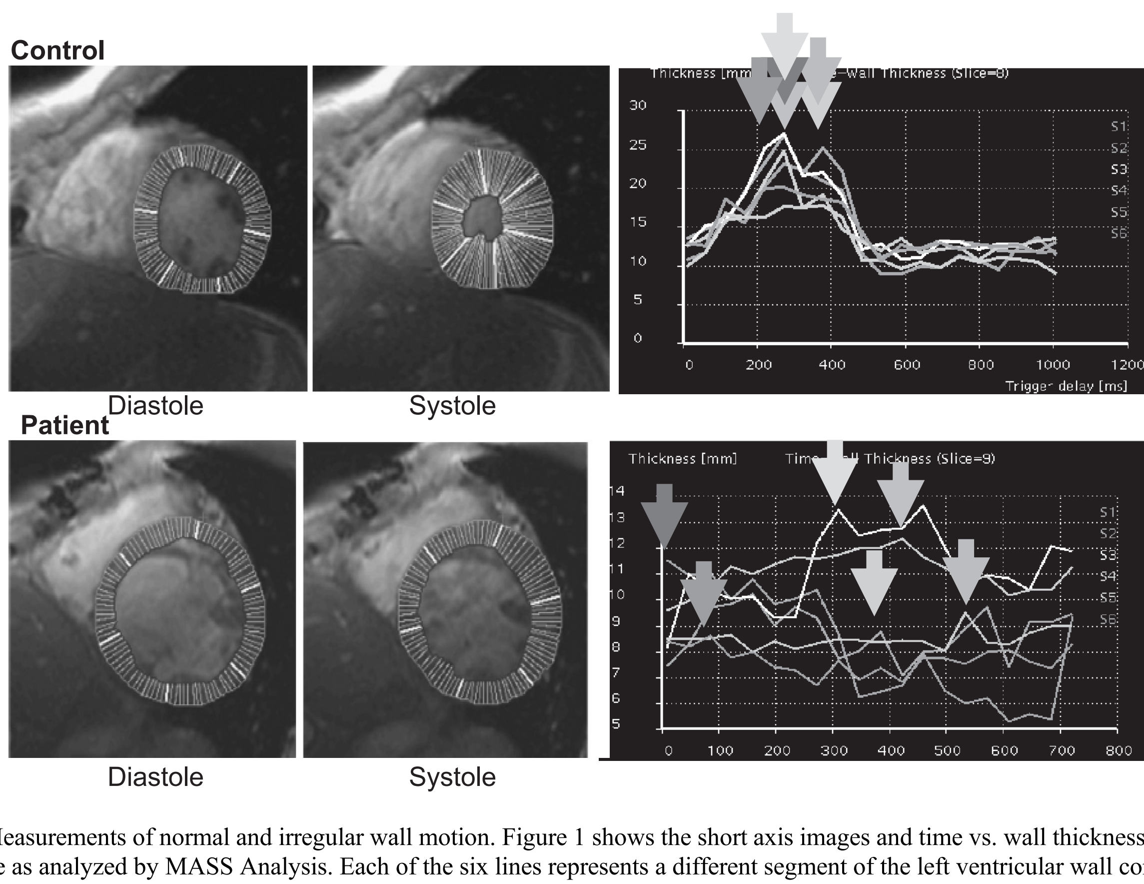

Figure 1. Measurements of normal and irregular wall motion. Figure 1 shows the short axis images and time vs. wall thickness graphs of the left ventricle as analyzed by MASS Analysis. Each of the six lines represents a different segment of the left ventricular wall contracting over a full cardiac cycle. Arrows mark the times of maximal contraction for each segment. In a typical control subject (top graph) all six segments of the ventricular wall reach their maximum thickness in systole at approximately the same time. However, in a dilated cardiomyopathy patient (bottom graph), each of the six segments of the ventricle reaches its maximum thickness in an uncoordinated manner over a longer period of time. Short axis slices of the left ventricle illustrate the difference in wall thickening between control subjects and patients with dilated cardiomyopathy.

Table 2. Asymmetry and asynchrony data: left ventricular function and wall motion analysis

Indices	Control (11)	HF (11)	p value (HF vs. controls)	CRT (5)	p value (CRT vs. controls)
Δmean (mm)	6.96 ± 1.7	2.88 ± 2.0	.00005	2.0 ± 1.2	.00003
Δmax (mm)	8.07 ± 2.4	4.37 ± 2.7	.003	.96 ± 2.01	.002
Δmin (mm)	5.45 ± 2.1	1.02 ± 1.3	.00002	0.51 ± 0.53	.000009
ΔΔ (mm)	2.62 ± 2.6	3.26 ± 2.5	.49	2.35 ± 1.9	.90
% ΔΔ	36.9 ± 38.1 (range 0.0–95.1)	189.8 ± 254.9 (range 24.4–945.0)	.07	125.8 ± 39 (range 66.8–172.0)	.003
(I_{sm})	10.9 ± 14.9 (range 0.0–46.4)	402.7 ± 427.3 (range 0.0–1438.1)	.01	988.5 ± 1676 (range 51.8–3972.0)	.26
(I_{sn})	3.2 ± 0.8 (range 2.0–4.5)	11.0 ± 4.2 (range 6.5–17.0)	.0001	8.6 ± 2.8 (range 6.0–12.5)	.01

This is a tabulation of mean values, standard deviations and p values of data from control, HF and CRT populations.

clockwise from the septal wall. Graphs of wall thickness over time for each of the six sections of the left ventricle were generated and wall thickening between the six segments were compared for measurements of asymmetry and asynchrony (Fig. 1).

Based upon the preliminary results summarized in Table 2, the index of I_{sm} was chosen as the primary indicator of asymmetry in heart contraction. I_{sm} was derived through the following terms: Δ_{mean}, $\Delta_{maximum}$, $\Delta_{minimum}$, $\Delta_{asymmetry}$, and %ΔΔ. Based upon analysis of time wall thickness graphs, each of the six segments of the left ventricular wall was

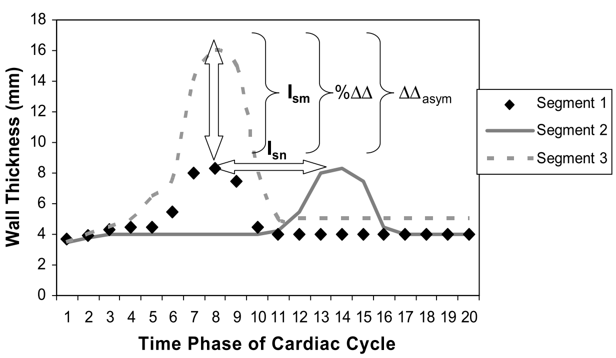

Figure 2. Asymmetry and asynchrony quantified from MASS analysis. Figure 2 plots myocardial wall thickness (mm) against time over a full cardiac cycle. Three segments of the left ventricle are represented in Fig. 1 of which is normally contracting (segment 3) and 2 of which are poorly contracting (segments 1 and 2). I_{sn} is measured as the difference in time phases between the point at which the first segment of the LV (segment 1) reaches its peak in contraction (time phase 8) and the point at which the last segment of the LV (segment 2) reaches its maximum in contraction (time phase 14). I_{sm}: Index of symmetry, the primary indicator of asymmetrical myocardial contraction; I_{sn}: Index of synchrony, the primary indicator of synchronous myocardial contraction; $\Delta\Delta_{asym}$: Difference in average wall thickness (mm) between the normally contracting and poorly contracting segments of the left ventricle (see methods section); % ΔΔ: Second order variable derived from $\Delta\Delta_{asym}$.—used to derive the I_{sm} value (see Methods section).

designated as either normally or poorly contracting. The average wall thickening (mm) for all normally contracting segments $\Delta_{maximum}$ and poorly contracting segments $\Delta_{minimum}$ of the left ventricle were measured from diastole to systole and the difference between the two values, $\Delta\Delta_{asymmetry}$ was calculated in millimeters. The average wall thickening between systole and diastole for all wall segments Δ_{mean} was also measured. Two second order variables %ΔΔ and I_{sm} were then determined by the formulas:

$$\%\Delta\Delta = (\Delta\Delta_{asymmetry}/\Delta_{mean}) * 100\% \qquad (1)$$

$$I_{sm} = (\%\Delta\Delta/\Delta_{minimum}) \qquad (2)$$

I_{sn} in left ventricular contraction was calculated from the spread of phases of the cardiac cycle over which segments of the LV achieved their maximum in contraction. Figure 2 provides a graphic representation of asymmetry and asynchrony values as defined by I_{sm} and I_{sn}.

All values of asymmetry were calculated for the mid slice of the heart, which was defined before data analysis to be the short axis slice of the heart at the level of the papillary muscle tips. Values of asynchrony were averaged over two short axis slices; the mid slice and the slice of heart apical to the mid slice.

2.3.2. *Determination of DCC by circumferential strain (Cs)*

MR tagging provides a means of tracking individual points on the myocardium over one cardiac cycle and directly images the in-plane motion of the heart wall (18). A tagging analysis program Harmonic Phase Magnetic Resonance Imaging (HARP) (Diagnosoft Inc., Maryland) was used to create graphs of circumferential strain (Cs) in controls vs. HF over one full cardiac cycle for apical slices of the left ventricle tagged by T2 weighted gradient echo sequences (20 images over the cardiac cycle). Analysis of spatial modulation of magnetization (SPAMM) by HARP allowed for measurement of strain in heart contraction. All control subjects also underwent MR tagging for circumferential strain analysis. However, data from only seven control subjects and 10 HF patients were amenable for analysis due to poor image

Table 3. Circumferential strain values in DCM vs. control

Circumferential strain (ECC)	Control (7)	HF (10)
Systolic strain (mid-ventricular slice)	−14.6 ± 3.3 range (−16.0 to −9.0)	−6.0 ± 5.3* range (−15 to 1)

This table shows the circumferential strain measurements of HF versus control subjects. This was found to have overlap and therefore was not a good marker in separating the two groups.
*P < 0.01 vs. control.

quality, software inaccuracies and irreproducibility. As a result of the tag fading in the latter half of the cardiac cycle, numerical data was only calculated for systole. Therefore, only 17 of the 27 tagged sequences were analyzed. The change in strain from the beginning of the cardiac cycle to its maxima in systole was measured by hand for one probe in the mid-ventricular septal region of the heart where the tags are best visualized and recorded as a measure of Cs (Table 3).

2.4. *Statistical analysis*

All results are presented as mean ± one standard deviation. Unpaired, two-tailed *t*-tests were used to calculate statistical significance between the HF and control populations. For the smaller CRT population (n = 5), the Mann-Whitney U ranking sum test was applied (19). Power calculations (20) were used to predict the number of patients it would take to achieve a p value of less than .01 between the control population and the HF and CRT groups.

3. Results

As defined in this study, normal heart contraction was synchronous as all segments of the left ventricular wall reached maximal contraction within an average span of three time phases (Table 2). Indices of asymmetrical and asynchronous myocardial contraction in representative control subjects were readily defined and relatively low. Mean values of asymmetrical contraction (I_{sm}) in controls were 40 times higher than in patients with heart failure and 90 times higher in patients qualifying for CRT (Fig. 3).

Indices of DCC in the control, HF, and CRT patient populations are summarized in Table 2. Measurements of I_{sm}, I_{sn}, and Cs in left ventricular myocardial contraction distinguished control subjects from HF patients with statistical significance ($p < .02$ and $p < .0002$, respectively). Of the three methods tested above, I_{sn} proved the most reliable, delineating control subjects from HF patients ($p < .0002$) and controls from CRT patients ($p < .02$) on all accounts with no overlap (Fig. 4). Measures of systolic Cs were not tested for statistical significant differences due to reasons already named. Though trials such as the 2002 MIRACLE trial have needed as many as 369 patients to reach

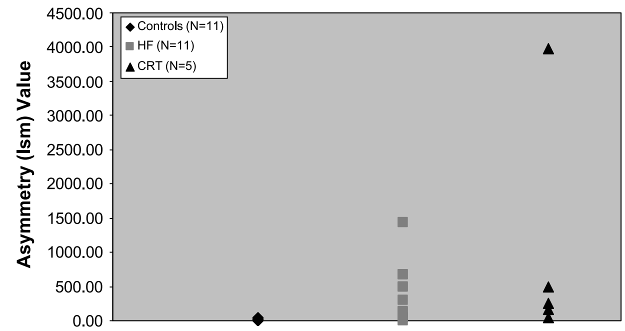

Figure 3. Measurement of asymmetry: %ΔΔ and I_{sm} in left ventricular contraction. This figure compares the mean asymmetry values of all three groups of the study population. Average values of I_{sm} are over 40 times higher in the HF patient group and 790 times higher in the CRT patient group than in the control population.

efficacy, (6) statistical power calculations revealed that our index, I_{sn}, could differentiate between CRT patients and control subjects at 100% power ($p < .05$), given the existing control and CRT population sizes (n = 11 and n = 5, respectively). This indicates that a much reduced number of patients will be needed for a future trial, if accurate, quantifiable measures of DCC are incorporated.

3.1. *Patient follow-up*

In order to establish a preliminary relationship between values of DCC and patient outcome upon receipt of CRT, follow-ups were conducted on members of the CRT patient group at a minimum of 3 months post surgery. Patients were interviewed on whether quality of life, evaluated based on an increase in patient mobility and improved disposition as in earlier published trials, had improved since pacing treatment and whether symptoms had been alleviated. Of the 5 patients interviewed, 4 reported that pacing had improved their lives for the better and that they were generally feeling more active

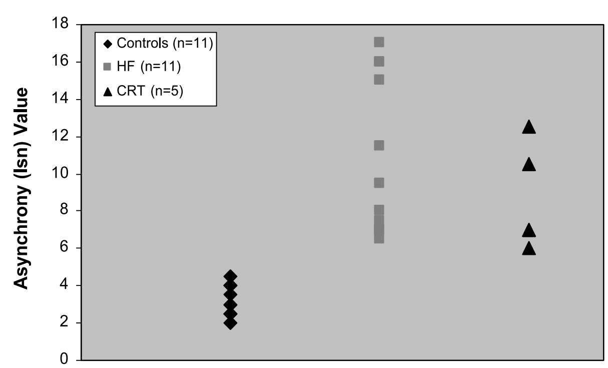

Figure 4. Measurement of asynchrony (I_{sn}) in left ventricular contraction. Figure 4 shows asynchrony results (as quantified with MASS Analysis) in the control, HF, and CRT patient groups. The graph shows that the synchrony indices of the HF and CRT patient groups are characterized by similar ranges, but are clearly distinct from the control values.

than before the surgery. One patient reported that his condition had not changed since receiving his pacemaker. This might be explained by his QRS of 133 ms, which barely qualified him with the current criteria, and his low asymmetry value of 51, almost 20 times lower than the mean of CRT patients. Though results cannot firmly establish a relationship between DCC and CRT response due to a low patient population, the 4 patients who reported improvement upon receipt of therapy generally did have higher asynchrony and asymmetry values than the patient who did not report improvement. Whilst anecdotal, these results reinforce the idea that indices of DCC may indeed be able to differentiate between responders and non-responders of CRT pacing.

4. Discussion

Our findings in this study defined novel quantitative criteria for DCC and confirmed that CMR is capable of distinguishing control subjects from HF patients using indices of DCC as defined by I_{sm}, I_{sn}, and Cs in left ventricular myocardial contraction. Of the three methods tested, I_{sm} and I_{sn} proved to be more consistent in differentiating between control subjects and HF patients. The use of more complex analysis by HARP to identify Cs proved to be a less reliable measure of distinguishing control subjects from heart failure patients, as demonstrated by the large overlap in measures of circumferential strain between control subjects and HF patients (Fig. 5). Recent improvements in commercially available HARP may prove beneficial.

Though measurements of DCC in control subjects and HF patients were easily distinguishable from one another (Table 2), DCC measurements in HF and CRT populations, as expected, had a large overlap. In terms of I_{sm} and I_{sn}, both the HF and CRT patient populations exhibited a wide range of values (HF $I_{sm} = 0.0–1438.1$, $I_{sn} = 6.0–17.0$; CRT $I_{sm} = 51.2–3972.0$, $I_{sn} = 6.0–12.5$) (Table 2). Though there was no statistically significant difference between HF and CRT populations in measured values of I_{sm} and I_{sn} (CRT vs. HF: $p = .5$ and $p = .2$, respectively), HF patients were characterized on average by a more asynchronous contraction pattern than the CRT patients. These results confirm that DCC is a universal feature of heart failure and that a number of HF patients who do not currently qualify for CRT exhibit somewhat more extreme signs of uncoordinated myocardial contraction than CRT candidates. Assuming that CRT treats heart failure by regulating myocardial contraction, our results indicate that these patients may also benefit from the procedure. Using the superior image quality provided by CMR, indices of DCC allow for the quantification of coordination in myocardial wall motion, which has previously been only poorly measurable (21).

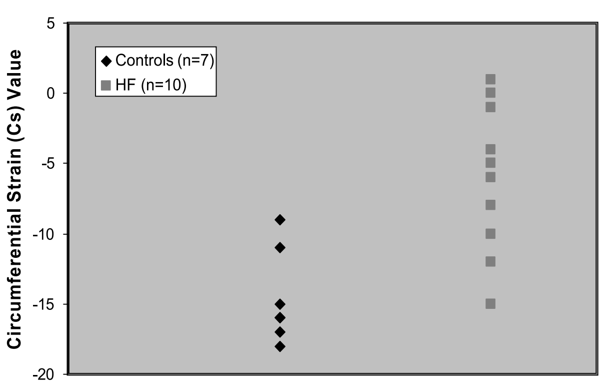

Figure 5. Circumferential strain in control vs. DCM. This figure shows the circumferential strain values of the control and HF populations. Only 57 controls were amenable to tagging analysis. Tagging values in the CRT population could not be analyzed by HARP due to poor image quality.

Currently, patients who are characterized by both a low EF (< 35%) and a broad QRS complex (>130 ms) are the only patients who qualify for CRT. According to the results of the MIRACLE Trial (2002), of these patients, an estimated 32 percent will not respond to the treatment. By adding DCC to the existing qualification criteria of CRT, we hope to eliminate the non-responders currently eligible for CRT and minimize their unnecessary surgery. At the same time, we hope to open the door to CRT candidacy for a number of potential CRT candidates who are characterized by significant measures of DCC but do not meet other CRT candidacy criteria such as a broad QRS complex >130 ms. A hypothesis to be tested in a future trial is whether patients with both a low EF (< 35%) and broad QRS complex (>130 ms) who also exhibit high levels of DCC will ultimately benefit from the treatment while other potential candidates will not. A study of the predictive value of DCC using clinical outcome after CRT is currently in progress. A final caveat is that because all HF patients were demonstrated to have high levels of DCC by the CMR criteria, we should consider whether these measurements are too sensitive to wall motion abnormalities and whether they need further refinement. Also, subjects were not randomized for this observational study. In a future trial, this would need to be added.

During the preparation of this manuscript, we found three publications (22–24), which deal with the assessment of cardiac resynchronization therapy through ultra sound tissue synchronization imaging and echocardiography. Ultrasound therefore provides similar novel tools in assessing asynchrony and outcome of cardiac resynchronization therapy as described here for MRI. The benefits of using tissue Doppler imaging or echocardiography is that it is safe for post-surgical evaluation, a limitation in MRI. However, the superior clarity of cardiac images and extraordinary precision with which asymmetry and asynchrony can be defined prior to surgery both indicate quantification of disordered wall motion by MRI to be worthy of further investigation.

4.1. *Limitations of the study*

A major limitation to this study was the pathology of heart failure itself. In several HF patients with extreme non-ischemic or dilated cardiomyopathy, minimal cardiac contraction and strain lead to relatively symmetrical contraction in myocardial segments of the left ventricle as no segments of the myocardium could contract sufficiently to exhibit asymmetrical wall motion. This resulted in an overlap in measurements of I_{sm} and Cs between several heart failure patients and control subjects. Furthermore, the spread of values obtained for I_{sm} in the CRT group was characterized by a non-Gaussian distribution. Because of this, regular statistical significance tests such as the two-tailed *t*-test, which relies upon a Gaussian distribution (25), could not be applied to the CRT values of I_{sm}. In these cases, the Mann-Whitney U ranking sum test (20) was used instead to test for statistical significance.

A second limitation of the study was a small CRT patient population. Though power calculations revealed that 5 CRT patients were sufficient to achieve efficacy, a detailed correlation between indices of DCC and heart failure improvement upon receipt of CRT could not be determined. Results indicate that patients characterized by severely asynchronous and asymmetrical myocardial contraction have a better chance of benefiting from the procedure than patients who do not, but a prospective study with an appropriate group size of CRT patients will need to be conducted to confirm the predicative value of DCC.

Due to the nature of the MRI exam, we were not able to re-examine the patients post-surgery which presented a major limitation in obtaining follow-up information of those patients. However, there are increasing efforts in the research of safety of MRI in implantable devices such as pacemakers (26). The results from such studies will soon prove to be very valuable to future CVMR studies.

5. Conclusions

Our findings in this exploratory study determined that disconjugate cardiac contractility (DCC) is a quantifiable measure of coordination of myocardial contraction easily measured in patients with heart failure by CMR. Indices of DCC are capable for distinguishing control subjects from patients with heart failure on all counts using measurements of I_{sm}, I_{sn}, and Cs in left ventricular wall motion. Assuming that DCC is a critical component of heart failure to be corrected by CRT, CMR may be efficacious in screening patients to improve the outcome of this valuable but costly therapy. Although TDI and echocardiography are less expensive, CVMR is also much less expensive than CRT, is universally available, and is likely to have an increasing role in all aspects of cardiac care.

6. Abbreviations

HF	heart failure
CRT	Cardiac Resynchronization Therapy
LV	Left Ventricle
EF	Ejection Fraction
ECG	Electrocardiogram
DCC	Disconjugate Cardiac Contractility
CMR	Cardiac Magnetic Resonance
I_{sm}	Index of Asymmetry
I_{sn}	Index of Asynchrony
Δmean	Average wall thickening
Δmax	Average wall thickening for normally contracting segments (of the LV)
Δmin	Average wall thickening for poorly contracting segments (of the LV)
Cs	circumferential strain
LVEDD	Left ventricular end-diastolic diameter
EDV	End diastolic volume
ESV	End systolic volume
SV	Stroke volume

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