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REVIEW

## Clinical applications of feature-tracking cardiac magnetic resonance imaging

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#### **Abstract**

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Cardiovascular diseases represent the leading cause of mortality and morbidity in the western world. Assessment of cardiac function is pivotal for early diagnosis of primitive myocardial disorders, identification of cardiac involvement in systemic diseases, detection of drug-related cardiac toxicity as well as risk stratification and monitor of treatment effects in patients with heart failure of various etiology. Determination of ejection fraction with different imaging modalities currently represents the gold standard for evaluation of cardiac function. However, in the last few years, cardiovascular magnetic resonance feature tracking techniques has emerged as a more accurate tool for quantitative evaluation of cardiovascular function with several parameters including strain, strain-rate, torsion and mechanical dispersion. This imaging modality allows precise quantification of ventricular and atrial mechanics by directly evaluating myocardial fiber deformation. The purpose of this article is to review the basic principles, current clinical applications and future perspectives of cardiovascular magnetic resonance myocardial feature tracking, highlighting its prognostic implications.

**Key words:** Left ventricular ejection fraction; Cardiac magnetic resonance; Cardiovascular disease; Strain; Feature-tracking

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Core tip: Cardiac magnetic resonance feature tracking analysis is progressively establishing its role as an



accurate tool to for quantitative evaluation of cardiovascular function by directly evaluating myocardial fiber deformation. Feature-tracking derived strain parameters are able to identify subtle myocardial abnormalities before overt clinical manifestation thus allowing early diagnosis of primitive cardiomyopathies, identification of cardiac involvement in systemic diseases, detection of drug-related cardiac toxicity as well as risk stratification and monitor of treatment effects in patients with heart failure of various etiology. The present article summarizes the basic principles, current applications and future perspectives of cardiovascular magnetic resonance myocardial feature tracking.

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#### INTRODUCTION

The evaluation of cardiac function has a pivotal role in diagnosis, risk stratification and assessment of treatment response in several cardiac disorders. Traditionally, left ventricular ejection fraction (LVEF) defined as the ratio between systolic output and end diastolic volume, assessed by various techniques including ventricular angiography, 2D- and 3D- echocardiography, cardiac single proton emission tomography, computed tomography and cardiac magnetic resonance (CMR), has represented the gold standard for evaluating cardiac function. Current international guidelines recommend the use of LVEF to assess the risk of sudden cardiac death (SCD) in patients with ischemic and non-ischemic cardiomyopathies, being patients with LVEF ≤ 35% at particular high risk and therefore referred for primary prevention implantable cardioverter defibrillator (ICD)[1]. Assessment of LVEF is also routinely recommended for familiar screening in patients affected by cardiomyopathies as well as to early detect cardiac involvement in systemic immune-mediated diseases or cardiac toxicity in patients undergoing cancer treatments<sup>[2-4]</sup>. However, in the last decade, new imaging modalities such as echo and CMR myocardial strain have progressively emerged as superior tools to evaluate global and regional myocardial mechanics, pointing out the limitations of LVEF especially in evaluating regional myocardial function and detecting early stage subclinical cardiac disorders<sup>[5]</sup>. In the present review we will focus on CMR feature tracking (CMR-FT) imaging of myocardial strain summarizing its basic principles, current clinical applications and future perspectives.

#### **GENERAL PRINCIPLES**

Myocardial strain is a deformation index defined as the

percentage change in dimension from a resting state (end-diastole) to the one achieved after contraction (end-systole)<sup>[6]</sup>. Considering a myocardial fiber, and being L0 its initial length in end-diastole and L1 its final length in end-systole, myocardial strain (S) can be defined as follow:

S = (L1 - L0)/L0 (Figure 1)

Due to the complex 3D architecture of the left ventricular (LV) musculature, systolic deformation takes place along several different directions. The evaluation of strain along each of these axes leads to the definition of different types of strain: (1) longitudinal strain (LS) represents the longitudinal shortening of the cardiac muscle, from the base to the apex, it is mostly determined by the longitudinally oriented muscle fibers in the subendocardial layer and is conventionally represented by a negative value due to systolic shortening; (2) circumferential strain (CS) is an expression of cardiomyocytes shortening along the LV circular perimeter, it is calculated in a short-axis view, it is mostly influenced by circumferentially oriented muscle fibers in the mid-wall and as well as LS it has a negative value due to systolic shortening; and (3) radial strain (RS) represents myocardial deformation toward the center of the ventricular cavity, and it is a measure of the LV thickening during systole and therefore is conventionally represented by a positive value.

Several other mechanical deformation parameters can be derived from strain analysis, of note: (1) Strain rate representing the "velocity" or rate at which the deformation occurs; and (2) LV torsion defined by the angle generated by the clockwise rotation of the basal segments and the counterclockwise rotation of the apex relatively to a stationary reference point conventionally located at a mid-ventricular level.

Myocardial CMR strain was initially investigated using tagging techniques in which magnetic labels (tags) are superimposed to the LV during cine imaging acquisition by radiofrequency spatial modulation of magnetization able to saturate parallel planes throughout the image, allowing the analysis of deformation of those lines throughout the cardiac cycle by post-processing semi-automated methods<sup>[7,8]</sup>. Soon after those initial experiences, new methods to evaluate myocardial strain based upon post-processing tracking of myocardial "features" overcame the need for prospective timeconsuming image acquisition protocols. Cardiac CMR-FT is able to detect and follow over time myocardial boundaries leading to a more automatized quantification of strain parameters<sup>[9]</sup>. From a technical point of view, the features tracked by CMR-FT are anatomic elements typically identified along the cavity-myocardial interface due to the high contrast resolution between blood pool and myocardium. Every feature is tracked across the cardiac cycle by specific algorithms searching the most similar image pattern on the following frame within a small window centered around the feature<sup>[10]</sup>. The endocardial and epicardial borders are usually manually traced in the end-diastolic phase, then the CMR-FT software automatically tracks the border across the



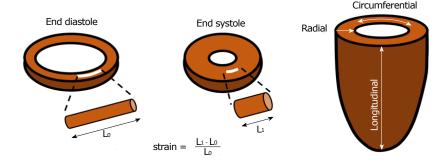


Figure 1 Schematic representation illustrating the physics principle beyond strain as well as the 3 main axes along which myocardial strain is calculated.

whole cardiac cycle. Global longitudinal strain (GLS) is derived by 2 or 3 long-axis steady state free precession (SSFP) cine images while global circumferential strain (GCS) and global radial strain (GRS) are derived from the short axis cine image stack. Several CMR-FT softwares are commercially available and can be directly applied to all CMR routine scans, as they work on standard SSFP cine images<sup>[11]</sup>.

#### FEATURE TRACKING NORMAL VALUES

Studies investigating CMR-FT strain values in healthy subjects have repeatedly demonstrated that global measurements are more reproducible compared to regional ones that are limited by partial volume effect with features fading/leaving the image plane between end diastole and end systole<sup>[12]</sup>. In particular, GCS seems to be less affected by inter-observer and intervendor variability compared to GRS and GLS in which the complex anatomy of the atrio-ventricular annular region may lead to poor tracking<sup>[11]</sup>. Strain values are affected by both gender and age but in general terms, values of circumferential and LS < -17% to -20% as well as values of RS > 25%-30% are considered within normal range<sup>[12-15]</sup>.

#### **CLINICAL APPLICATIONS**

#### Ischemic heart disease

Both global and regional strain parameters as well as dyssynchrony indices have been widely investigated in patients with ischemic heart disease. The main areas of interest include the correlation between regional strain parameters and infarct characteristics, the impact of strain parameters on long-term LV remodeling and the ability of strain to predict long-term clinical outcomes and to identify inducible ischemia<sup>[16]</sup>.

All strain parameters are impaired in infarcted territories with strain values inversely related to the infarct size and infarct transmurality<sup>[17,18]</sup>. Compared to wall motion abnormalities, segmental strain values allows a more accurate discrimination between areas of subendocardial from transmural infarction and non-infarcted remote zones with only certain limitations in

the early post-infarct phase due to the coexistence of edema, inflammatory cells, hemorrhage and viable and necrotic myocardial fibers<sup>[17,19-21]</sup>. Among all strain parameters, CS has shown to be the more accurate in assessing infarct extension and has also demonstrated its superiority to conventional LVEF determination in identifying subtle impairments of LV contractile function<sup>[19,22,23]</sup>.

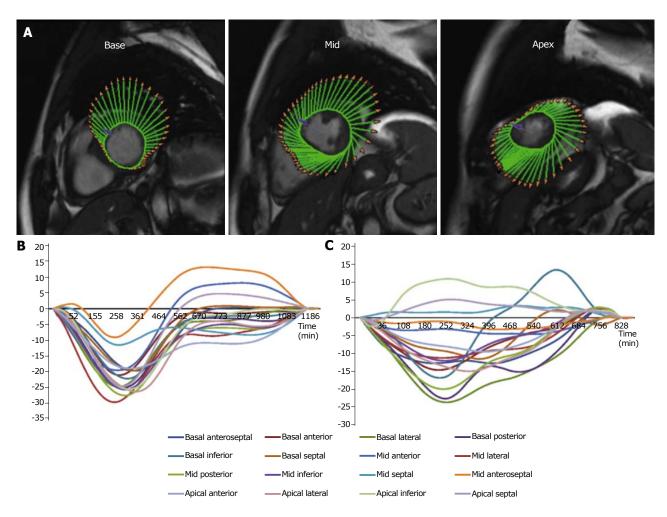
Data concerning the capability of strain analysis to predict adverse LV remodeling after acute myocardial infarction are debating<sup>[24-26]</sup>. In a study including 74 patients who underwent CMR within 4 d after successfully reperfused ST-elevation myocardial infarction (STEMI), a cut-off value of -19.3% for GCS appeared as accurate as late gadolinium enhancement (LGE) extension to identify patients with recovered LVEF (≥ 50%) at 6-mo follow-up while no significant correlation was determined using GLS<sup>[24]</sup>. In another study including 164 STEMI who underwent CMR a median of 3 d after the acute event, CS was not inferior to the area of LGE in predicting segmental functional improvement/ normalization after a median of 9-mo<sup>[25]</sup>. Conversely, Shetye et al<sup>[26]</sup> have recently failed to demonstrate a correlation between baseline strain parameters and the development of adverse LV remodeling after 4-mo follow-up among 65 patients with STEMI who underwent CMR within 3 d post-reperfusion, even though a good correlation was found between strain parameters, baseline LVEF and infarct size.

The correlation between scar extent determined by LGE imaging and long-term risk of major adverse cardiac events (MACE) after myocardial infarction is well known<sup>[27]</sup>. However, it has been recently pointed out how various CMR-FT derived parameters maybe able to accurately predict long-term clinical outcomes (Table 1). In a large study by Gavara *et al*<sup>[28]</sup>, the prognostic value of CMR-FT was investigated among 323 patients who underwent CMR one week after a STEMI. During a median follow-up of 36-mo, all strain parameters were correlated to the incidence of a composite endpoint including cardiac death, readmission for heart failure and reinfarction. However, after adjustment for baseline and CMR variables, GLS (HR 1.21; 95%CI: 1.11-1.32; P < 0.001) was the only independent predictor of MACE. In

Table 1 Principal studies analyzing the prognostic role of cardiac magnetic resonance-feature tracking in patients with ischemic and non-ischemic cardiomyopathy

Cost many of s <sup>[2]</sup> , 2017         323         HD (recent SIDM)         GSS (architer) dends from the and sendenction         17         GIS (2.2)         Seno (monthard)           Absolute of s <sup>[2]</sup> , 2015         1.89         HD (recent SIDM)         Cackbowsecular death, shorted SCD and Sendenction on heart dishare 1.8         2         Cackbowsecular death, shorted SCD and Sendenction on heart dishare 1.8         8 mon (monthard)         85 mon (monthard)           Nisetions of s <sup>[2]</sup> , 2017         1.39         HBD (recent SIDM)         More heart dishare 1.8         2         Accid (1.16)         85 mon (monthard)           Nisetions of s <sup>[2]</sup> , 2017         1.30         HBD (recent SIDM)         More heart dishare 1.8         2         Accid (1.16)         85 mon (monthard)           Niset s <sup>[2]</sup> , 2017         1.30         HBD (recent SIDM)         More heart dishare 1.8         2         Accid (1.16)         85 mon (monthard)           Niset s <sup>[2]</sup> , 2018         1.10         HBD (recent SIDM)         More heart dishare 1.8         2         Accid (1.16)         85 mon (monthard)           Niset s <sup>[2]</sup> , 2018         1.10         HBD (recent SIDM)         More heart dishare 1.8         3         Accid (1.16)         85 mon (monthard)           Niset s <sup>[2]</sup> , 2018         1.10         HBD (recent SIDM)         More heart dishare 1.8         Accid (1.16)         Accid (1.16)	Reference	No. of patients	Herat disease	Parameters analyzed	Outcome	Occurrence of outcome, %	Independent predictors of the outcome event (HR)	Follow-up duration
180         IHD (recent STBM)         GCS         Cardiovascular death, aborted SCD and LIGE         22         GCS (1.16)           130         IHD (recent STBM)         Mechanical dispersion of cardiovascular death, aborted SCD and LIGE         Amount and aborted SCD and Light         Amount LS (5.44)         Amount LS (5.45)         Amount LS (5.44)         Amount LS (5.44)         Amount LS (5.44)         Amount LS (5.44)         Amount LS (5.44)	Gavara <i>et al<sup>[28]</sup>, 2017</i>	323	IHD (recent STEMI)	GLS GCS GRS n. segments with altered LS n. segments with altered CS LVEF LGE	Cardiac death, readmission for heart failure and reinfarction	17	GI.S (1.21)	36 mo (median)
130         IHD (recent STEMI)         Mechanical dispersion (1.39)         Cardiovascular death, aborted SCD and LUNE         Cardiovascular death, heart transplant         12         Mechanical dispersion (1.39)           210         IDCM         GIS         Composite of cardiac death, heart transplant         12         GIS (1.27)           Abean LS         Abean LS         Mean LS         Abean LS (3.44)         Mean LS (3.44)           146         IDCM         LVBF         Composite of cardiac death, heart transplant         16         Long axis strain (1.28)           147         IHD + IDCM         LONG axis strain         Composite of cardiac death heart transplant         16         LONG axis strain (1.28)           1012         IHD + IDCM         GIS         All-cause death         20         GIS (2.33)           1012         IHD + IDCM         GIS         All-cause death         173         GIS (1.89)           102         IDCM         GIS         Composite of cardiac death and heart         25         ILGE (4.73)           102         GIS         CGS         Composite of cardiac death and heart         25         ILGE (4.73)	Nucifora $et  al^{[29]}$ , 2018		IHD (recent STEMI)	MVO GCS LVEF LGE MVO	Cardiovascular death, aborted SCD and hospitalization for heart failure	22	GCS (1.16)	95 mo (median)
210         IDCM         GLS         Composite of cardiac death, heart transplant         12         GLS (1.27)           GCS         Mean LS         Mean LS         Mean LS (3.44)         Mean LS (3.44)           Mean CS         Mean RS         Mean LS (3.44)         Mean LS (3.44)           Mean RS         Mean RS         Mean LS (3.44)         Mean LS (3.44)           Mean RS         Mean RS         LOFE         LOFE           LOFE         LORE         LORE         LORE (3.44)           LOFE         LORE         All-cause death         LORE (3.51)           LOFE         LORE         All-cause death         LORE (3.53)           LOFE         LORE         All-cause death         LORE (3.53)           LORE         LORE         All-cause death         LORE (3.53)           LORE         LORE         All-cause death         LORE (4.73)           LORE         CGS         transplant         25         LGE (4.73)           LORE         LORE         LORE         LORE         LORE         LORE	Muser <i>et al</i> <sup>[30]</sup> , 2017	130	IHD (recent STEMI)	Mechanical dispersion LVEF LGE MVO	Cardiovascular death, aborted SCD and hospitalization for heart failure	20	Mechanical dispersion (1.39)	95 mo (median)
146         IDCM         Long axis strain         Composite of cardiac death, heart transplant         16         Long axis strain (1.28)           LVEF         LVEF         and aborted SCD         LVEDV/BSA (1.01)           LVEDV         LGE         All-cause death         20         GLS (2.35)           LVEF         LVEF         All-cause death         13         GLS (2.35)           1012         IHD + NICM         GLS         All-cause death         13         GLS (1.89)           LVEF         LGE         Composite of cardiac death and heart         25         LGE (4.73)           GCS         LWFF         CRS         LGE (4.73)           LWF         LWF         LWF         LGE (4.73)	Buss et al <sup>iss</sup> , 2015	210	IDCM	GCS GCS GRS Mean LS Mean RS LVFF	Composite of cardiac death, heart transplant and aborted SCD	12	GLS (1.27) Mean LS (5.44)	5.3 yr (median)
470         IHD + IDCM         GLS         All-cause death         20         GLS (2.35)           LVEF         LVEF         LVEF (0.95)         LVEF (0.95)           1012         IHD + NICM         GLS         All-cause death         13         GLS (1.89)           LVEF         LGE         Composite of cardiac death and heart         25         LGE (4.73)           GCS         transplant         25         LGE (4.73)           LVEF         LGE         LGE	Riffel et a $l^{[\infty]}$ , 2016	146	IDCM	Long axis strain LVEF LVEF LVEF LVEF	Composite of cardiac death, heart transplant and aborted SCD	16	Long axis strain (1.28) LVEDV/BSA (1.01) LGE (2.51)	$4.3 \pm 2.0 \text{ yr}$
1012         IHD + NICM         GLS         All-cause death         13         GLS (1.89)           LVEF         LGE         Composite of cardiac death and heart         25         LGE (4.73)           GCS         transplant         GRS         LGE (4.73)           LVEF         LGE         LGE (4.73)	Romano <i>et al</i> <sup>[37]</sup> , 2017	470	IHD + IDCM	GIS LVEF LGE	All-cause death	20	GLS (2.35) LVEF (0.95)	4 yr (median)
172         IDCM         GLS         Composite of cardiac death and heart         25         LGE (4.73)           GCS         transplant           GRS         LVEF           LGE         LGE	Romano <i>et al</i> <sup>[38]</sup> , 2018	1012	IHD + NICM	GLS LVEF LGE	All-cause death	13	GLS (1.89)	4.4 yr (median)
	Pi et $a^{[39]}$ , 2018	172	IDCM	GLS GCS GRS LVEF LGE	Composite of cardiac death and heart transplant	25	LGE (4.73)	47 mo (median)

IHD: Ischemic heart disease; STEMI: ST-elevation myocardial infarction; GLS: Global longitudinal strain; GCS: Global circumferential strain; GRS: Global strain; LVEP: Left ventricular ejection fraction; LGE: Late gadolinium enhancement; MVO: Microvascular obstruction; SCD: Sudden cardiac death; IDCM: Idiopathic dilated cardiomyopathy; LVEDV: Left ventricular end diastolic volume; BSA: Body surface area.



**Figure 2** Example of left ventricular endocardial feature tracking. A: Example of tracking of the endocardial border of the left ventricle (LV) on basal, mid, and apical short-axis, steady-state free-precession images using an dedicated feature-tracking software. The software automatically calculates the circumferential strain of each myocardial segment; B: Example of a patient with homogenous LV contraction; C: Example of a patient with extreme regional heterogeneity of myocardial contraction. Reprinted with permission from Muser *et al*<sup>30</sup>].

particular, MACE rate was higher in patients with a GLS  $\geq$  -11% (22% vs 9%; P = 0.001). In the same study, those results were also validated in an external cohort of 190 STEMI patients confirming both the higher incidence of MACE in patients with a GLS  $\geq$  -11% (34% vs 9%; P < 0.001) and the independent predictive value of GLS (HR 1.18; 95%CI: 1.04-1.33; P = 0.008)<sup>[28]</sup>. These findings were subsequently confirmed and extended by our group. We have reported GCS to be significantly and independently associated to the occurrence of a composite endpoint including cardiovascular death, aborted SCD and hospitalization for heart failure (HR 1.16; 95%CI: 1.07-1.25; P<0.001) during a median followup of 95 mo in a population of 180 patients admitted for a first STEMI and who underwent CMR imaging a median of 8 d following the index event<sup>[29]</sup>. We have also reported how early assessment (median of 7 d after STEMI) of LV mechanical dispersion defined as the standard deviation of the time-to-peak CS of the LV segments, is significantly and independently related to the occurrence of MACE (HR 1.39, 95%CI: 1.20-1.62, P < 0.001) (Figure 2)<sup>[30]</sup>.

Due to its high sensitivity in identifying contractile

function compared to visual assessment of wall motion abnormalities or determination of changes in LVEF, CMR-FT has recently been proposed to evaluate inducible ischemia and establishing contractile reserve in patients with chronic ischemic heart disease<sup>[31,32]</sup>. Schneeweis et al[33] have demonstrated how in 25 patients with suspected or known coronary artery disease, undergoing dobutamine stress CMR, CS during high doses of dobutamine, was able to identify segments supplied by a vessel of > 70% narrowing with a sensitivity of 75% and specificity of 67% using a strain threshold of -33.2%<sup>[33]</sup>. In the same study, the authors also showed how impairment of CS already occurred at intermediatedoses of dobutamine allowing an earlier detection of inducible ischemia compared to visual assessment<sup>[33]</sup>. In another study including 15 patients undergoing viability assessment by low-dose dobutamine stress, there was no response to dobutamine in dysfunctional segments with scar transmurality > 75% while dysfunctional segments without scar showed improvement either in subendocardial and subepicardial GCS as well as in GRS. In particular, GCS improved in all segments up to a transmurality of 75% while GRS improved in

segments with < 50% transmurality and remained unchanged above 50% transmurality<sup>[32]</sup>.

#### Idiopathic dilated cardiomyopathy

The presence and extension of scar represents a negative prognostic factor in patients with idiopathic dilated cardiomyopathy (IDCM), as well<sup>[27]</sup>. Unfortunately, no clear evidence of LGE can be found in up to 60% of these patients making extremely important to identify new potential predictors to further stratify individual risk beyond simple LVEF<sup>[34]</sup>. A study including 210 patients with IDCM found GLS to be an independent predictor of MACE including cardiac death, heart transplantation and aborted SCD (HR 1.27, 95%CI: 1.06-1.52, P < 0.02) during a median followup of 5.3-years regardless to the baseline LVEF and the presence of LGE, while no significant association was found with GCS and GRS<sup>[35]</sup>. Another study including 146 patients affected by IDCM, investigated the value of "long axis strain" defined as the distance between the epicardial border of the LV apex and the midpoint of a line connecting the origins of the mitral valve leaflets in end-systole and end-diastole in predicting a combination of cardiac death, heart transplantation and aborted SCD and found that the ratio between LV enddiastolic volume/body surface area (HR 1.01, 95%CI: 1.00-1.02, P = 0.04), the presence of LGE (HR 2.51, 95%CI: 1.02-6.19, P = 0.04) as well as long axis strain (HR 1.28, 95%CI: 1.07-1.52, P < 0.01) were all independent predictors of MACE<sup>[36]</sup>. The incremental value of CMR-FT strain was subsequently confirmed in a series of 470 patients of whom 140 with IDCM, in which was described an independent correlation of GLS (HR 2.35, 95%CI: 1.81-3.06, P < 0.001) and LVEF (HR 0.95, 95%CI: 0.91-0.99, P = 0.038) with the risk of death during a median follow-up of 4-years regardless to the presence and extension of LGE<sup>[37]</sup>. In a large multicenter study including 1012 patients with both ischemic heart disease and non-ischemic cardiomyopathy, GLS was an independent predictor of all-cause mortality over LVEF and presence of LGE in the all cohort (HR 1.89, 95%CI: 1.55-2.07, P < 0.001) as well as in the ischemic (HR 1.95, 95%CI: 1.48-2.58, *P* < 0.001) and non-ischemic (HR 2.14, 95%CI: 1.56-2.91, P < 0.001) subgroups<sup>[38]</sup>. The above results have not been confirmed by a recent study including 172 patients with IDCM and moderately to severely reduced LVEF (<40%). In this study, neither GLS, GCS or GRS were correlated with the risk of death or heart transplant during a median follow-up of 47-mo, while presence of LGE and serum sodium were the only independent predictors of the outcome event (Table 1)<sup>[39]</sup>.

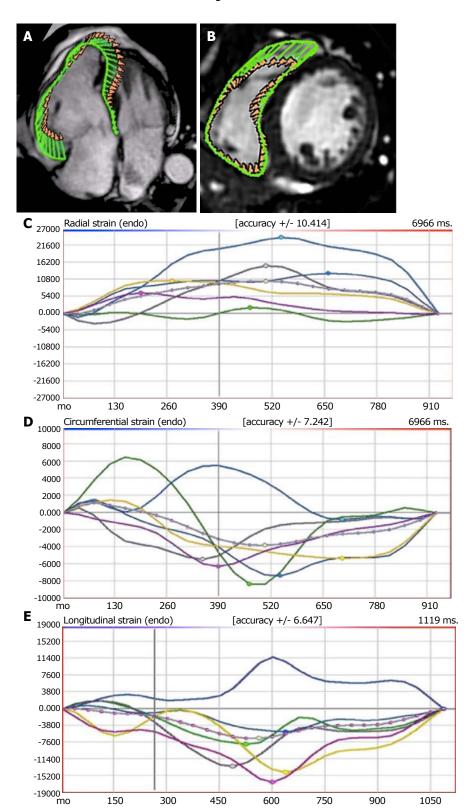
#### Other forms of non-ischemic cardiomyopathy

Myocardial strain has proven to be a valuable tool in early identification, staging and risk stratification of several forms of non-ischemic cardiomyopathy. In patients with hypertrophic cardiomyopathy (HCM), a direct correlation between strain parameters and the presence of LGE has been repeatedly proven, being the presence of replacement fibrosis associated to a reduction in CS<sup>[40,41]</sup>. However, intramural strain has demonstrated to be reduced in more hypertrophied segments compared to the non-hypertrophied ones regardless to the presence of LGE, proving myocardial scarring not to be the only determinant of regional contractile dysfunction<sup>[42]</sup>.

Global and regional right ventricular (RV) strain parameters have shown to be impaired in patients with overt arrhythmogenic right ventricular cardiomyopathy (ARVC) regardless to RV dimensions and function<sup>[43,44]</sup>. In a study comparing RV strain parameters in 32 patients matching the task force criteria for ARVC but with no or only minor CMR criteria, to 32 patients with idiopathic RV outflow tract premature ventricular contractions and 32 healthy volunteers, we found that RV GLS, GCS and GRS were all significantly reduced in the ARVC group. In particular, a RV GLS > -23.2% was able to identify 88% of ARVC patients without definite CMR criteria showing the incremental value of CMR-FT over conventional CMR imaging in early detection of the disease (Figure 3)<sup>[45]</sup>.

In the same direction, strain parameters have shown to be impaired in patients with acute or previous myocarditis and preserved LVEF regardless to the presence of LGE confirming the higher sensitivity of CMR-FT in identifying contractile impairment at a subclinical stage<sup>[46,47]</sup>. In patients affected by LV non-compaction, we have found that subclinical impairment of myocardial deformation, occurs early in the natural history of the disease, being noticeable since the pediatric age with e reduction in all global strain parameters while overt LVEF reduction tends to manifest only later in adulthood<sup>[48]</sup>. In this regard, CMR-FT may be used to early detect cardiac involvement in systemic diseases or during administration of drugs with potential cardiotoxic effects. Monitoring of cardiotoxicity during cancer therapies is currently recommended by echocardiographic evaluation of LVEF and a decline in the LVEF is needed in order to decide to suspend/modify therapy<sup>[3]</sup>. Nakano et al[49] have demonstrated how both GLS and GCS were significantly reduced after 6 mo of therapy with trastuzumab in 9 women treated for breast cancer. Changes in global strain parameters were correlated with changes in LVEF, however their predictive value on the development of heart failure needs to be proven<sup>[49]</sup>.

Some preliminary data have shown that parameters derived from CMR-FT such as LV rotational indices are disease specific and therefore maybe be used to differentiate between cardiomyopathies. We compared LV twist and untwist rates between 20 patients with cardiac amyloidosis (CA) to 20 patients with HCM and 20 healthy controls showing how both peak LV twist and peak LV untwist rates were significantly impaired in patients with CA while peak LV twist rate was significantly increased in patients with HCM compared





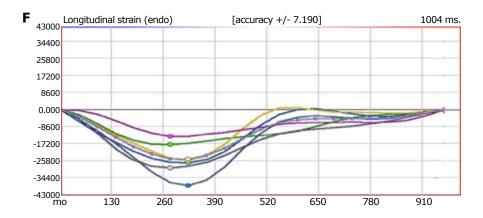


Figure 3 Example of endocardial feature tracking of the right ventricle. A: Tracking of the endocardial border of the right ventricle (RV) on a 4-chamber steady-state free precession image using a dedicated feature-tracking software; B: Example of tracking on the mid-section of a short-axis view; C: Radial; D: Circumferential; E: Longitudinal strain patterns of a patient with arrhythmogenic RV cardiomyopathy; F: Example of the RV longitudinal strain pattern of a healthy subject. Reprinted with permission from Prati et al<sup>45</sup>.

to controls. Patients with HCM also presented a preserved peak LV untwist rate. The time to peak LV untwist rate was significantly prolonged both in CA and HCM compared to healthy subjects<sup>[50]</sup>.

#### Congenital heart diseases

The follow-up of grown-up patients with congenital heart diseases (CHD) is usually based upon echocardiography, however, the poor acoustic window due to previous multiple surgical procedures or coexistence of other anatomical deformities as well the complex anatomy of repaired congenital defects represents some major limitations. For this reason, CMR imaging is increasingly becoming the imaging modality of choice for evaluation of CHD. In these terms CMR-FT derived parameters may have important clinical implications in evaluating surgical results and in early detection of complications. In patients with repaired tetralogy of Fallot (TOF), for example, a significant impairment of all global strain parameters has been described in those patients who experienced death or sustained ventricular tachycardia compared to those of similar age who did not experienced outcome events<sup>[51]</sup>. An improvement in LV GCS and GLS within 6 mo after transcatheter implantation of a pulmonic valve has also been documented in patients with repaired TOF and clinically relevant residual pulmonary regurgitation/stenosis after initial corrective surgery<sup>[52]</sup>. Moreover, RV GLS has been related to clinically relevant variables such as exercise capacity and oxygen consumption in 28 patients with repaired TOF<sup>[53]</sup>. Those findings have been further confirmed in a recent large prospective series of 372 repaired TOF patients, in which LV GCS and RV GLS independently predicted death, aborted SCD or documented ventricular tachycardia during a median follow-up of 7.4 years<sup>[54]</sup>. Similar findings have been found in 15 patients who underwent Fontan palliative intervention; GCS and GLS of the single ventricle both correlated with New York Heart Association class and peak oxygen uptake on cardiopulmonary exercise

test<sup>[55]</sup>. Among patients with successful repaired coarctation of the aorta and preserved LVEF, impairment of GLS but not GCS or GRS has been associate to coexistence of LV hypertrophy<sup>[56]</sup>.

#### Cardiac resynchronization therapy

A significant proportion of cardiac resynchronization therapy (CRT) patients may fail to reach an adequate response in terms of LVEF and HF status improvement and those patients are commonly addressed as "nonresponders". Increasing efforts have been made in the last years to better select optimal candidates to CRT but also to identify the best site for LV lead implantation in order to maximize the chances of response<sup>[57,58]</sup>. For this reason, CMR has emerged as a valuable technique been able to noninvasively evaluate LV activation patterns and dyssynchrony $^{[30,45,59]}$ . Taylor *et al* $^{[60]}$  have demonstrated that CRT implantation guided by determination of segments with latest mechanical activation (defined by time to peak systolic CS) and absence of LGE is able to improve LV reverse remodeling as well as long term survival and reduce the risk of hospitalizations for heart failure. From a practical point of view, CMR derived information on scar, dyssynchrony and coronary venous anatomy maybe integrated with fluoroscopy or 3D-electroanatomical mapping systems to realtime guide LV lead placement during interventional procedures[61,62].

#### Atrial physiology

The application of CMR-FT techniques to the left atrium (LA) have shown to accurately characterize LA physiology compared to simple global measures such as atrial volume, area and atrial ejection fraction<sup>[63]</sup>. It has been advocated that changes in LA function precede the development of heart failure in several cardiac disorders. The development of LA dysfunction due to increased LV stiffness associated to the presence of replacement or diffuse ventricular fibrosis has been described as a potential marker of early diastolic disfunction<sup>[64]</sup>.



Recent findings highlighted how there may be disease specific patterns of LA dysfunction. Patients with HCM, for example, present an increased contractile function compared with healthy controls. On the other side, patients with diastolic dysfunction and preserved LVEF have reduced atrial contractility<sup>[65]</sup>. There is some evidence that quantitative measurement of LA function may also have prognostic implications as impairment in LA strain has shown to precede development of heart failure in the general population and to improve risk stratification for cerebrovascular events in patients with atrial fibrillation<sup>[66,67]</sup>.

#### Special populations

Analysis of CMR-FT strain parameters has been applied to patients with isolated bicuspid aortic valve (BAV) (*i.e.*, without aortic stenosis, aortic regurgitation or aortic dilatation)<sup>[68]</sup>. Interestingly, patients with "clinically normal" BAV have significant impairment of LV systolic and diastolic myocardial mechanics; furthermore, the impairment of LV systolic mechanics observed in BAV patients is independently related to the congenital abnormality of aortic valve itself. The authors postulated that the observed intrinsic impairment of LV contractility may accelerate the ominous LV remodeling pathways occurring after the development of significant aortic valve dysfunction, possibly explaining the described premature occurrence of congestive heart failure in BAV patients compared with the general population<sup>[69]</sup>.

CMR-FT strain analysis has been also applied to study the adult consequences of pre-term birth. Pre-term birth interferes with the normal in-utero development of the heart, potentially leading to abnormally remodeled left ventricles. In a large study, 102 subjects have been prospectively followed since pre-term birth (gestational age =  $30.3 \pm 2.5$  wk) to the age of 20-39 years when they underwent a CMR study. Compared to 132 subjects born at term of similar age, preterm individuals had increased LV mass proportional to the degree of prematurity and short LVs with small internal diameters and a displaced apex. Interestingly, even if LVEF was similar to subjects born at term, both longitudinal systolic (peak strain, strain rate, and velocity) and diastolic (peak strain rate and velocity) function as well as rotational (apical and basal peak systolic rotation rate, net twist angle) parameters were significantly lower<sup>[70]</sup>. Similar findings have been described by the same group affecting the RV. Pre-term subjects had smaller RV with bigger RV mass with the severity of differences proportional to gestational age. Moreover, differences in RV function were greater than those reported for the LV as subjects born pre-term had significantly lower RV ejection fraction (RVEF) with 6% of them having clear RV systolic dysfunction. In agreement with the lower RVEF, RV GLS and peak systolic strain rate were also significantly lower compared to subjects born at term<sup>[71]</sup>. The clinical implications of these findings on the potential development of overt cardiomyopathies need

further prospective evaluation.

#### CONCLUSION

In the last years a growing amount of evidence suggests that study of cardiac function by strain analysis can accurately detect cardiac disorders at a subclinical level, improve risk stratification of patients with various cardiac conditions and potentially monitor treatment effect. In this setting, the use of CMR-FT may represent an easy and fast tool due to its applicability to routinely acquired SSFP cine images by dedicated software without need for upfront use of specific protocols. Further technical improvements able to achieve a higher level of accuracy and reproducibility in the assessment of measures and more standardization between different vendors are still required to make CMR-FT ready to use in routine clinical practice.

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