ADF by CMR was directly associated with oxygen pulse, but not with any other EST parameters. ADF by echo was directly associated with physical working capacity and percentage of predicted maximum oxygen consumption in the group that reached a maximal EST (**Table 1**).

Continued efforts have been made to better define determinants of outcomes in TOF. Restrictive RV physiology has been shown to be a presumably protective phenomenon, associated with less severe PI by echo and superior exercise performance (2). In this study we report that ADF is commonly identified, with no significant difference in detection rate by echo and CMR and is associated with worse PI but with superior exercise performance in the echo ADF group that achieved a maximal exercise test. Similar to previous studies, ADF was commonly observed on CMR and echo, regardless of CMR sedation status, therefore even though the method used to detect ADF differs between echo and CMR, these imaging modalities can be potentially interchangeable for this assessment (1).

We found that ADF was directly associated with severity of PI, RV dilation, and stroke volume. These findings are in contrast with the initial description of restrictive physiology as a protective phenomenon associated with smaller cardiac silhouettes and less severe PI, but similar to others (1,3). We postulate that ADF occurs in the setting of an ineffective native pulmonary valve that fails to impede antegrade flow during atrial contraction, in the presence of greater pulmonary arterial bed capacitance. Finally, ADF was associated with better oxygen consumption in the ADF plus echo patients that achieved a maximal test, similarly to the original report on ADF, but contrary to others (1,4). This finding merits future study since our study does not allow us to infer whether ADF results in better oxygen consumption or, alternatively, better oxygen consumption reflects a particular hemodynamic state in patients with more severe PI that allows for ADF. Furthermore, we were limited by the absence of respiratory tracing on echo and cannot directly compare our findings to others.

In conclusion, ADF is associated with worse PI and possibly with better aerobic capacity. ADF likely represents a complex phenomenon that reflects a combination of greater RV volume, atrial contractility, and pulmonary arterial bed capacitance, with decreased RV compliance.

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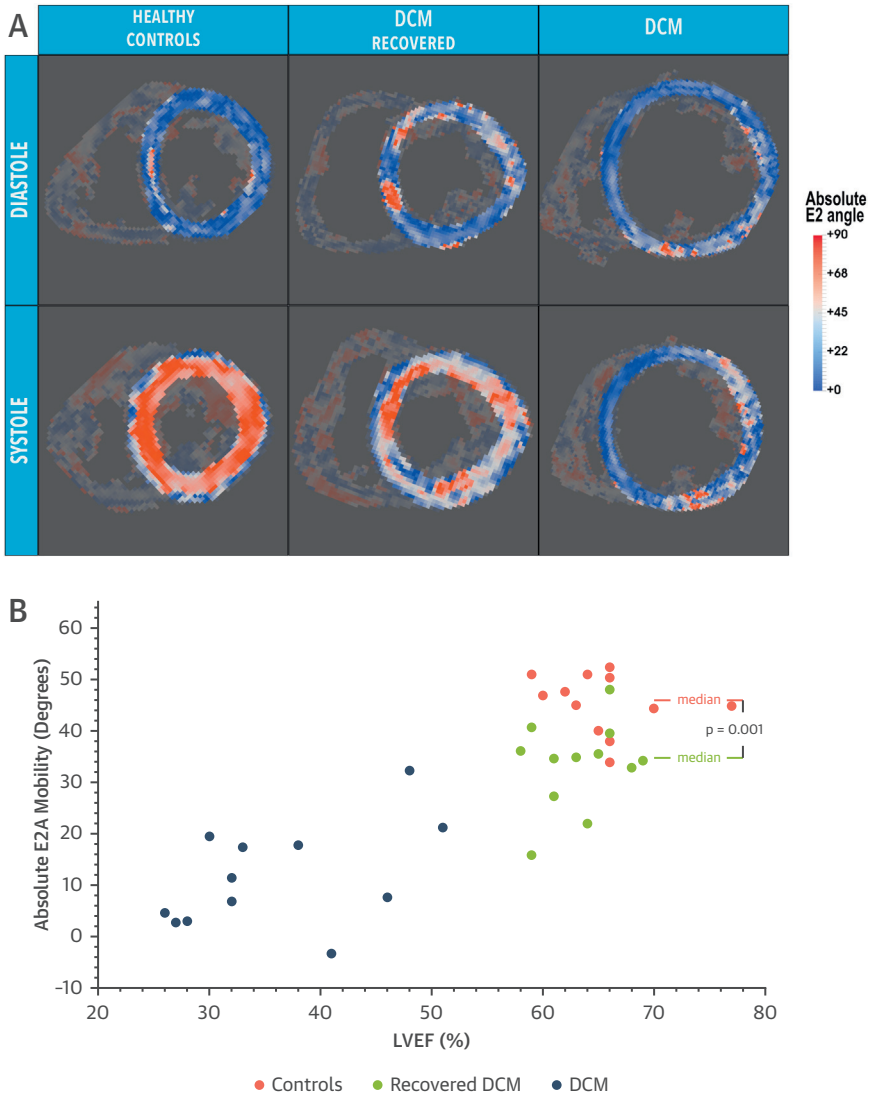
Diffusion Tensor Cardiovascular Magnetic Resonance of Microstructural Recovery in Dilated Cardiomyopathy

Diffusion tensor cardiovascular magnetic resonance (DT-CMR) evaluates myocardial microstructure, using helix angle (HA) and absolute angulation of the second eigenvector (E2A) to assess cardiomyocyte and sheetlet orientation respectively. In health, sheetlets align more wall-parallel in diastole and more wall-perpendicular in systole. However, in dilated cardiomyopathy (DCM), with left ventricle (LV) dilatation and systolic dysfunction, sheetlet mobility is reduced (1). In one-third of idiopathic DCM patients, LV size and ejection fraction (EF) can improve with pharmacological and device therapy (2). We performed DT-CMR to assess microstructural recovery in recovered DCM (R-DCM).

Twelve DCM patients, 12 R-DCM patients, and 12 controls were studied using standard criteria (1). In R-DCM, LV size and EF were within normal indexed ranges, and evidence of prior LV dilation and reduced EF by CMR or echocardiogram was required. Patients with a clinical diagnosis of

FIGURE 1 **Microstructural Abnormalities Persist in R-DCM**



(A) Diastolic E2A shows predominance of wall-parallel values **(blue)**. Systolic maps show more wall-perpendicular higher E2A **(red)** in controls, but not to the same extent in R-DCM and even less in DCM. **(B)** E2A mobility plotted against LVEF. The DCM group have reduced EF and E2A mobility. Although R-DCM and controls have similar EFs (64% [60% to 66%] vs. 66% [62% to 66%]; p = 0.32), they are distinguishable by the lower E2A mobility in R-DCM. Peak radial strain was 0.42 (0.32 to 0.45) in R-DCM, more than DCM (0.18 [0.11 to 0.28]; p = 0.002), but less than controls (0.55 [0.50 to 0.67]; p = 0.009). DCM = dilated cardiomyopathy; E2A = angle of the second eigenvector; EF = ejection fraction; LVEF = left ventricular ejection fraction; R-DCM = recovered dilated cardiomyopathy.

idiopathic, genetic, or familial R-DCM were included. DT-CMR was performed as previously described (1). The Mann-Whitney test compared groups with Bonferroni correction.

Groups were well-balanced for age and sex. Median (interquartile range) age was 61 (44 to 66) years in R-DCM, 54 (41 to 59) years in DCM, and 54 (43 to 60) years in controls. In DCM, LV size and mass were increased (end-diastolic volume 154 ml/m^2 [123 to 201 ml/m^2] and 88 g/m^2 [72 to 115 g/m^2]), whereas EF was reduced (33% [29% to 45%]). LV volume and mass were not significantly different between R-DCM (82 ml/m^2 [75 to 91 ml/m^2]; 62 g/m^2 [54 to 72 g/m^2]) and controls (80 ml/m^2 [70 to 87 ml/m^2]; 66 g/m^2 [59 to 74 g/m^2]).

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Controls EF was 66% (62% to 66%). In R-DCM, baseline EF was 27% (25% to 38%) and current EF was 64% (60% to 66%). For the imaged slice midwall, late gadolinium enhancement was present in 9 of 12 DCM patients and 5 of 12 R-DCM patients. All patients were in New York Heart Association functional class 1.

Myocyte orientation HAs were negative epicardially, near circumferential mesocardially, and positive HAs endocardially and similar across groups. Median (interquartile range) diastolic E2A in R-DCM was 25° (22 to 30°), which was similar to DCM (19° [16 to 27°]; $p = 0.11$) and controls (20° [15 to 29°]; $p = 0.24$). However, systolic E2A in R-DCM was 59° (52 to 66°), significantly greater than DCM (35° [22 to 38°]; $p < 0.0001$), but lower than controls (65° [63 to 71°]; $p = 0.01$). E2A mobility was 35° [29 to 39°] in R-DCM, greater than DCM (9.6° [3.4 to 19°]; $p < 0.0001$), but less than controls (46° [41 to 51°]; $p = 0.001$) (**Figure 1**).

Myocardial contractile recovery is complex and multifactorial, with increasing focus on different patients groups with reverse remodeling, remission and full recovery (3). Clinically, R-DCM is based upon structural, biochemical, and symptomatic improvement with improved LV size and LVEF >40% to 50%. However, recovered patients can still demonstrate abnormal biomarker levels, subclinical dysfunction, and substantial cardiac hospitalizations and heart failure symptoms (4,5). In this study, all R-DCM patients described symptomatic and structural improvement with entirely recovered LV size and EF within reference ranges. However, systolic E2A and sheetlet mobility remained significantly reduced in R-DCM compared with controls indicating persistent microstructural abnormalities in R-DCM, despite normalization of LV size and EF. E2A mobility in R-DCM was greater than in DCM, however, implying a variable microstructural measure that alters along the disease trajectory. This suggests a potential role of DT-CMR in the assessment of DCM recovery/remission and predicting patients at risk of relapse, and reinforces the ability of DT-CMR to provide insight into LV dynamics beyond the limitations of EF.

Limitations of DT-CMR (1) include low spatial resolution, affecting measurements in thin walls. In this cross-sectional study with retrospective analysis of DCM diagnosis, patient heterogeneity remains. This includes disparity between echo and CMR derived EF. Further work in DCM of extrinsic origin such as myocarditis may yield different microstructural findings.

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Mismatch Between Diameter Stenosis and Plaque Atheroma Volume

Challenging Glagov Phenomenon?

We eagerly read the report by Lee et al. (1) that found slower rate of total and noncalcified percentage of atheroma volume (PAV) progression and a faster progression of calcified PAV in statin-taking patients without history of coronary artery disease who underwent coronary computed tomography angiography at an interscan interval ≥2 years when compared with statin-naïve patients. To gain an understanding of the results, some questions remain unanswered.

This report conflicts with other progression/regression trials for the following reasons: 1) the