institutions that provide ACVI training, marked differences exist in the duration of training and modalities offered.

Given these findings, we believe that the ACC and the ACVI professional societies should work together to augment the quality and availability of ACVI training. First, they should jointly develop a more comprehensive multimodality imaging training statement with structured ACVI training pathways exclusive of the basic exposure to ACVI required for general cardiology fellowship. This will help to ensure high quality of all ACVI training programs and trainees. After standardization of training curricula, the ACC and the ACVI professional societies should dedicate the necessary resources to define and measure the value that ACVI experts add to clinical, research, and educational programs. Doing so will incentivize more institutions to invest in ACVI training. Ultimately, improvements in the quality and availability of ACVI training will help to establish ACVI as a board-certified subspecialty.

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REFERENCES

1. Narula J, Chandrashekhar YS, Dilsizian V, et al. COCATS 4 Task Force 4: training in multimodality imaging. J Am Coll Cardiol 2015;65:1778–85.

2. Advanced Imaging Training Program Database. Available at: http://www.acc.org/membership/sections-and-councils/imaging-section/training-resources. Accessed November 12, 2015.

Longitudinal Strain as Risk Factor for Outcome in Pediatric Dilated Cardiomyopathy
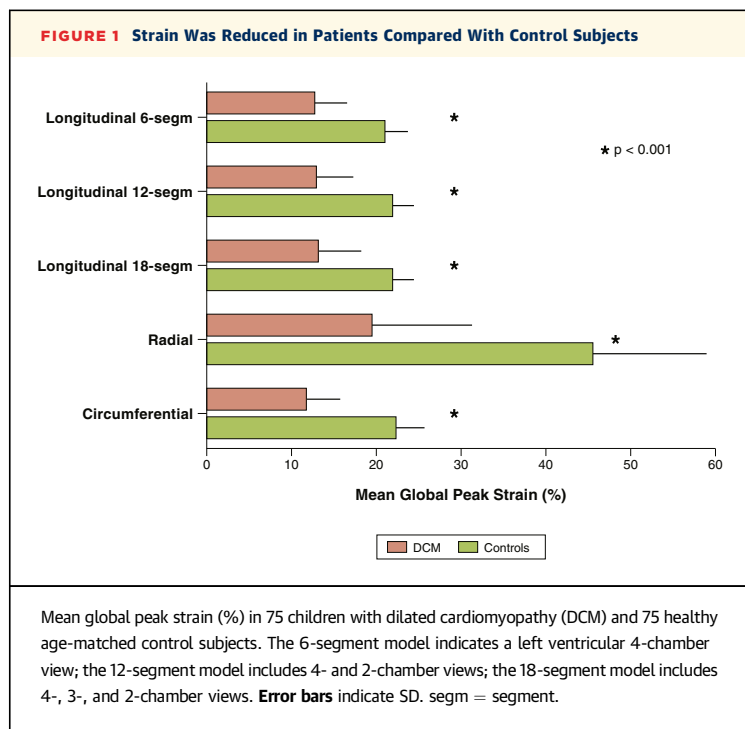
In adults with dilated cardiomyopathy (DCM), it has been demonstrated that global longitudinal and circumferential strain have value in addition to left ventricular ejection fraction (LVEF) in predicting the risk of mortality, heart transplantation, and hospitalization for worsening heart failure [1]. Measures that predict disease progression and outcome in children with DCM are needed. Therefore, we aimed to evaluate the predictive value of left ventricular (LV) global peak strain in the outcome in children with DCM.

We prospectively included 75 children (younger than 18 years of age) with DCM (left ventricular end-diastolic dimension [LVEDD] z-score ≥2 for body surface area and fractional shortening [FS] ≤25% on echocardiography) from 7 academic pediatric cardiology centers. Available data for 75 healthy age-matched controls were used [2]. A complete 2-dimensional echocardiographic study was performed in a standardized way; measurements (LV dimension, function, and speckle-tracking echocardiography) were performed in a core echocardiography laboratory, as previously described [2]. The mean age of all subjects was 7.5 ± 6.3 years. Patients were included at a median time of 1 year (interquartile range [IQR]: 0.1 to 4.0 years) after DCM diagnosis; the mean LVEF was $33 \pm 11\%$, and the mean LVEDD z-score was 5.1 ± 3.0. The mean LV global peak strain in all views was significantly reduced compared with that in control subjects (Figure 1). No specified regions could be identified as more affected than others because all segments were worse in patients than in control subjects ($p < 0.001$). The 6-segment model (longitudinal 4-chamber view) was feasible in 99% of the patients and the short-axis view in 92% of the patients. The 12-segment model (including the 4- and 2-chamber views) was feasible in 85% and the 18-segment model (including the 4-, 2-, and 3-chamber views) in 64%. The mean global peak strain of the 6-, 12-, and 18-segment models were comparable ($13 \pm 4\%$, $13 \pm 4\%$, and $13 \pm 5\%$, respectively). Interobserver variability and intraobserver variability of longitudinal and circumferential strain were good (intraclass correlation coefficients: 0.88 to 0.91) and of radial strain was moderate (0.63).

The median follow-up from echocardiography until an endpoint or censoring was 21 months (IQR: 16 to 31 months); 10 patients (13%) reached a primary endpoint: 8 underwent heart transplantation and 2 died. Using univariable Cox regression analysis, we found that lower mean global longitudinal peak strain of the 4-chamber was significantly associated with a higher risk of an endpoint; each percentage of decrease in strain gave a 1.23 times higher risk of death or heart transplantation (hazard ratio [HR]: 0.81 per percentage of increase in strain, $p = 0.04$). LVEF (HR: 0.96; $p = 0.19$), FS (HR: 0.95; $p = 0.28$), LVEDD z-score (HR: 1.12; $p = 0.22$), and circumferential peak strain (HR: 0.83; $p = 0.07$) were not significantly associated

FIGURE 1 Strain Was Reduced in Patients Compared With Control Subjects

Mean global peak strain (%) in 75 children with dilated cardiomyopathy (DCM) and 75 healthy age-matched control subjects. The 6-segment model indicates a left ventricular 4-chamber view; the 12-segment model includes 4- and 2-chamber views; the 18-segment model includes 4-, 3-, and 2-chamber views. **Error bars** indicate SD. segm = segment.

with outcome, nor were the 12-segment model (HR: 0.89; p = 0.17) and the 18-segment model (HR: 0.93; p = 0.50) significantly predictive of outcome.

This is the first study to report that, in pediatric DCM, LV global longitudinal strain was predictive of death and heart transplantation. In addition, circumferential strain tended to be significant and had good reproducibility, suggesting that it may be of interest for future studies. According to its feasibility and comparable strain results, in addition to its prognostic value, we advise using the 6-segment model for longitudinal strain.

Our findings are in accordance with adult results (1). Until now, pediatric studies have mainly focused on measures at diagnosis, whereas FS and LVEDD at diagnosis were predictive of death and heart transplantation (3). In the present study, patients had a median time of 1 year after diagnosis. Therefore, our results indicate that in the follow-up of pediatric DCM, LV global longitudinal peak strain may be used to predict outcome.

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REFERENCES

1. Motoki H, Borowski AG, Shrestha K, et al. Incremental prognostic value of assessing left ventricular myocardial mechanics in patients with chronic systolic heart failure. J Am Coll Cardiol 2012;60:2074–81.

2. Klitsie LM, Roest AAW, van der Hulst AE, et al. Assessment of intra-ventricular time differences in healthy children using two-dimensional speckle-tracking echocardiography. J Am Soc Echocardiogr 2013;26:629–39.

3. Alvarez JA, Orav EJ, Wilkinson JD, et al. Competing risks for death and cardiac transplantation in children with dilated cardiomyopathy. Circulation 2011;124:814–23.

High-Output Heart Failure in Sickle Cell Anemia

We read with great interest the article by Niss et al. (1) in the March 2016 issue of *iJACC*. These investigators describe an abnormal mitral inflow pattern, left atrial enlargement, and pulmonary hypertension in a cohort of patients with sickle cell anemia. In the absence of a definitive pathogenesis, they suggest the potential for an underlying restrictive cardiomyopathy as an explanation for the underlying Doppler mitral inflow abnormalities and diastolic dysfunction.

We propose an alternative explanation for the cardiac structural and hemodynamic parameters noted. Sickle cell anemia is characterized by significant anemia, along with compensatory intra-medullary and extramedullary hematopoiesis. This may be associated with increased metabolic demand, peripheral vasodilation, and a resultant high-cardiac output state (2). One can also speculate that in addition to anemia, increased metabolic demand and shunting from exuberant bone marrow hematopoiesis may also contribute to a high-cardiac output state. This condition has been reported in other hematologic diseases such as multiple myeloma (3). High-output heart failure may occur if systemic