Commentary: Who *doesn't* have abnormal myocardial strain?

Katerina A. Boucek, MD,^a and William M. DeCampi, MD, PhD^b

From the ^aDivision of Pediatric Cardiology, Department of Pediatrics, The Medical University of South Carolina, Charleston, SC; and the ^bDepartment of Clinical Sciences, The Heart Center, Arnold Palmer Hospital for Children, College of Medicine, University of Central Florida, Orlando, Fla.

Disclosures: Authors have nothing to disclose with regard to commercial support.

Received for publication March 9, 2019; accepted for publication March 11, 2019; available ahead of print April 17, 2019.

Address for reprints: William M. DeCampi, MD, PhD, The Heart Center, Arnold Palmer Hospital for Children, 83 W Miller St, Orlando, FL 32813 (E-mail: William.decampi@ucf.edu).

J Thorac Cardiovasc Surg 2019;158:520-2

0022-5223/\$36.00

Copyright © 2019 by The American Association for Thoracic Surgery

<https://doi.org/10.1016/j.jtcvs.2019.03.022>

A 252-lead ECG vest for noninvasive mapping (Cardiolsight; Medtronic, Minneapolis, Minn).

Central Message

Abnormal myocardial strain seems very common after repair of numerous congenital anomalies. The reasons and implications are unclear. Serial measurements and advanced technology will help clarify.

See Article page 509.

During the past decade, systolic myocardial strain (deformation) has become a popular way to detect early left and right ventricular systolic dysfunction. Myocardial strain (with speckle-tracking echocardiography or, more recently, feature-tracking cardiac magnetic resonance imaging) can be measured locoregionally to produce a “map” of systolic activity that reproduces the complex way in which the ventricle contracts. Deviations from the “normal” pattern reflect inefficient pump action and, ipso facto, ventricular systolic dysfunction. Deviations from the normal can correspond to (1) regional decrease in the magnitude of strain, (2) “mechanical dyssynchrony,” deviations from the normal temporal sequence of mechanical activation of each region or (3) decrease in regional rate of change of strain over the cardiac cycle (“strain rate”). These findings may be associated, in turn, with abnormal electrical conduction patterns. Abnormalities may be detected even in the presence of normal ejection fraction, and in the absence of imaging evidence of perfusion defects and myocardial scarring.

In this issue of the *Journal*, Schäfer and colleagues¹ report the results of an observational study of 29 patients who underwent either the Ross or Ross-Konno procedure.¹ At a median of 10 years of follow-up, patients underwent feature-tracking cardiac magnetic resonance imaging imaging for myocardial strain analysis. Relative to patients with normal cardiac anatomy, one or both operative groups had (1) reduced global longitudinal strain (both Ross and Ross-Konno), (2) increased interventricular mechanical dyssynchrony (both Ross and Ross-Konno), (3) increased left ventricular intraventricular mechanical dyssynchrony (Ross-Konno), and (4) correlation between intraventricular mechanical dyssynchrony and QRS duration on 12-lead electrocardiogram (Ross-Konno). All patients had normal functional status. Schäfer and colleagues¹ suggest that strain measurements might be a better way to follow long-term ventricular performance in these patients.

The study of Schäfer and colleagues¹ is a valuable contribution to our understanding of the myocardial functional status of patients who have undergone Ross and Ross-Konno procedures. Are their findings surprising? There are hundreds of articles touting abnormal strain magnitude or strain patterns in a myriad of congenital and acquired cardiac disorders. In congenital heart disease, decreased global longitudinal strain has been cited in long-term follow-up after repair of ventricular septal defect,² tetralogy of Fallot,³ coarctation,⁴ hypoplastic left heart syndrome (after stage 1 repair),⁵ tricuspid atresia (after Fontan operation),⁶ transposition of the great arteries,⁷ and aortic valve disease.⁸ In fact, Menting and associates⁹ found abnormal apical right ventricular longitudinal strain in a cohort of 51 patients at a median follow-up of 35 years after atrial septal defect repair! For patients with one or more levels of left ventricular outflow tract obstruction treated with a Ross or Ross-Konno procedure, the possible mechanisms of persistent abnormal strain are intuitively obvious: preoperative myocardial hypertrophy with subendocardial ischemia and microfibrosis; intraoperative injury to myocardium, coronary arteries, or conduction system; and postoperative subtle abnormalities in afterload and abnormal right ventricular function with adverse right-left ventricular coupling. Finally, one cannot rule out genetic determinants

FIGURE 1. Longitudinal strain (A) and strain rate (B) versus preload recruitable stroke work (PRSW) with corresponding regression lines and 95% confidence intervals. Regression functions with regression coefficients and *P* values. *NS*, Not significant; *SL*, longitudinal strain; *SrL*, longitudinal strain rate. From: Dahle and colleagues.¹⁰ Used with permission.

in some cases. Whatever the underlying cause of the problem, it is interesting that the problem persists a decade after repair.

One major problem with measuring and tracking strain magnitude alone is that, like ejection fraction or fractional shortening, it is preload dependent. So, how does one know that one is measuring strain under the same conditions across a cohort of patients? In 2016, Dahle and coworkers¹⁰ demonstrated that *strain rate*, not strain, correlated best with preload recruitable stroke work and with left ventricular maximal rate of change in pressure—load-independent measures of cardiac contractility and therefore a more reliable measure along time and across a patient population (Figure 1). Studying strain rate might comprise a logical extension of the current work of Schäfer and colleagues.¹

Few, if any, articles report a set of entirely normal strain metrics in any patient cohort with any form of congenital or acquired cardiac disorder. If all the positive findings are reliable, they collectively imply that most, if not all, forms of heart disease or their treatments are associated with long-term systolic dysfunction. The outstanding question is what we do with this information. Studies are beginning to appear that report serial measurements of strain as a tool to predict outcomes. For example, Colquitt and coworkers⁵ used serial measurements of right ventricular strain during a 6-month period to predict mortality in hypoplastic left heart syndrome. Others (including Schäfer and colleagues) have used strain measurements to assess mechanical dyssynchrony (as described previously). In this article, Schäfer and colleagues¹ evaluated intraventricular mechanical dyssynchrony by the variance of the time of maximum strain among all measured myocardial regions. A better measure may be the deviation of the activation sequence from that of the “normal” activation sequence. Computational models now exist to determine the efficiency of kinetic energy transfer to blood flow, given the mechanical activation sequence of the myocardium.

Finally, some studies (including this one) have examined the association between mechanical and electrical dyssynchrony to attempt to determine to what extent intrinsic conduction abnormalities are responsible for mechanical dyssynchrony. Although the QRS duration and morphology crudely measure electrical dyssynchrony, determination of the actual activation sequence by electrophysiologic mapping would be better. Like myocardial strain, this can now be performed noninvasively. By recording signals from roughly 250 electrocardiographic electrodes placed on the patient (Figure 2), a computational algorithm can localize and calculate approximately 1500 unipolar epicardial electrograms to map the electrical activation sequence.¹¹ Statistical techniques exist to correlate the electrical map with the mechanical strain map.

The work of Schäfer and colleagues¹ certainly suggests that “all is not right” with the myocardium long after the Ross and Ross-Konno operations. Their study lays down another stepping stone toward the full use of the

FIGURE 2. A 252-lead electrocardiographic vest for noninvasive mapping (CardioInsight; Medtronic, Inc, Minneapolis, Minn).

aforementioned metrics, techniques, and technologies to deepen further our understanding of the long-term consequences of repaired congenital heart disease.

References

1. Schäfer M, Browne LP, von Alvensleben JC, Mitchell MB, Morgan GJ, Ivy DD, et al. Ventricular interactions and electromechanical dyssynchrony after Ross and Ross-Konno operations. *J Thorac Cardiovasc Surg.* 2019;158:509-17.
2. Kwok SY, Yeung SS, Li VW, Cheung YF. Ventricular mechanics after repair of subarterial and perimembranous VSDs. *Eur J Clin Invest.* 2017;47.
3. Jing L, Haggerty CM, Suever JD, Alhadad S, Prakash A, Cecchin F, et al. Patients with repaired tetralogy of Fallot suffer from intra-and inter-ventricular cardiac dyssynchrony: a cardiac magnetic resonance study. *Eur Heart J Cardiovasc Imaging.* 2014;15:1333-43.
4. Li VW, Cheung YF. Right ventricular myocardial motion and deformation in adolescents and young adults after repair of coarctation of the aorta. *Echocardiography.* 2015;32:797-804.
5. Colquitt JL, Loar RW, Morris SA, Feagin DK, Sami S, Pignatelli RH. Serial strain analysis identifies hypoplastic left heart syndrome infants at risk for cardiac morbidity and mortality: a pilot study. *J Am Soc Echocardiogr.* February 22, 2019 [Epub ahead of print].
6. Singh GK, Cupps B, Pasque M, Woodard PK, Holland MR, Ludomirsky A. Accuracy and reproducibility of strain by speckle tracking in pediatric subjects with normal heart and single ventricular physiology: a two-dimensional speckle-tracking echocardiography and magnetic resonance imaging correlative study. *J Am Soc Echocardiogr.* 2010;23:1143-52.
7. Grotenhuis HB, Cifra B, Mertens LL, Riessenkampff E, Manliot C, Seed M, et al. Left ventricular remodeling in long-term survivors after the arterial switch operation for transposition of the great arteries. *Eur Heart J Cardiovasc Imaging.* 2019;20:101-7.
8. Deng MD, Wei X, Zhang XL, Li XD, Lui GY, Zhu D, et al. Changes in left ventricular function in patients with aortic regurgitation 12 months after transcatheter aortic valve implantation. *Int J Cardiovasc Imaging.* 2019;35:99-105.
9. Menting ME, van den Bosch AE, McGhie JS, Cuypers JA, Witsenburg M, Geleijnse ML, et al. Ventricular myocardial deformation in adults after early surgical repair of atrial septal defect. *Eur Heart J Cardiovasc Imaging.* 2015;16:549-57.
10. Dahle GO, Stangeland L, Moen CA, Salminen PR, Haaverstad R, Matre K, et al. The influence of acute unloading on left ventricular strain and strain rate by speckle tracking echocardiography in a porcine model. *Am J Physiol Heart Circ Physiol.* 2016;310:H1330-9.
11. Ehrlich MP, Laufer G, Coti I, Peter M, Andreas M, Stix G, et al. Noninvasive mapping before surgical ablation for persistent, long-standing atrial fibrillation. *J Thorac Cardiovasc Surg.* 2019;157:248-56.