Right Ventricular Systolic and Diastolic Function as Assessed by Speckle-Tracking Echocardiography Improve With Prolonged Isolated Left Ventricular Assist Device Support

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ABSTRACT

Background: Right ventricular (RV) failure is a major cause of morbidity and mortality after left ventricular assist device (LVAD) implantation. Whether RV function deteriorates with prolonged LVAD support is unknown. Speckle-tracking echocardiography provides a sensitive, noninvasive, reproducible, and quantitative assessment of RV systolic and diastolic function.

Methods: Echocardiograms were retrospectively reviewed from before and after implantation of a Heartmate II LVAD. Speckle-tracking analysis was performed to measure RV longitudinal systolic strain, strain rate, and diastolic strain rate for each patient at baseline and over discrete time periods after LVAD implantation.

Results: Seventeen patients were included in the analysis, with an average follow-up after LVAD implantation of 234 ± 125 days. RV systolic strain improved in 15 patients, decreasing from −7.4 ± 2.3% to −9.7 ± 3.3% after LVAD ($P = .026$). Systolic strain rate improved in 11 patients, decreasing from −0.67 ± 0.25%/s to −0.96 ± 0.36%/s ($P = .011$). RV diastolic strain rate improved in 12 patients, increasing from 0.70 ± 0.33%/s to 1.02 ± 0.40%/s ($P = .016$).

Conclusions: Chronic LVAD support improves RV systolic and diastolic function in LVAD patients who did not require an RV assist device. Speckle-tracking echocardiography may offer a noninvasive technique for identifying and monitoring improvements in RV function in LVAD patients. (*J Cardiac Fail 2014;20:498−505*)

Key Words: Mechanical circulatory support, heart failure, myocardial recovery, ventricular remodeling.

The assessment of right ventricular (RV) function among patients supported with a left ventricular assist device (LVAD) is of critical importance. RV failure is one of the leading causes of morbidity and mortality after LVAD implantation.[1] In addition, as an LVAD primarily unloads the left ventricle (LV), it is unknown whether underlying RV pathology progresses over time, leading to deterioration in RV function among patients chronically supported with a LVAD. This is of increasing clinical relevance as a growing number of patients are being supported with LVADs for a prolonged period of time—either as destination therapy or while waiting for an extended period of time for a transplant owing to limited donor organ availability. Furthermore, very little data exist to show whether RV myocardial contractility deteriorates during extended LVAD support and contributes to the exceedingly low rates of myocardial recovery observed in clinical practice.[2]

In spite of its clinical importance, the noninvasive assessment of RV systolic and diastolic function is exceedingly challenging. Transthoracic echocardiography is the most commonly used imaging modality for assessing RV function; however, the RV's crescent shape and anterior location limit the ability to reproducibly and quantitatively assess RV function.[3] Qualitative assessments of RV size and function can be subjective and prone to variability, so quantitative assessments such as tricuspid annular plane systolic excursion (TAPSE), 2-dimensional RV dimensions, RV ejection fraction (EF), and estimations of RV systolic pressure calculated from tricuspid regurgitation jet are commonly used to assess RV function. However, such assessments provide limited information regarding RV diastolic function, produce

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Manuscript received November 22, 2013; revised manuscript received March 7, 2014; revised manuscript accepted April 22, 2014.

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Funding: Dr Ambardekar is supported by a Scientist Development Grant (13SDG17030001) from the American Heart Association and by the Boettcher Foundation's Webb-Waring Biomedical Research Program.

See page 505 for disclosure information.

1071-9164/$ - see front matter

http://dx.doi.org/10.1016/j.cardfail.2014.04.017

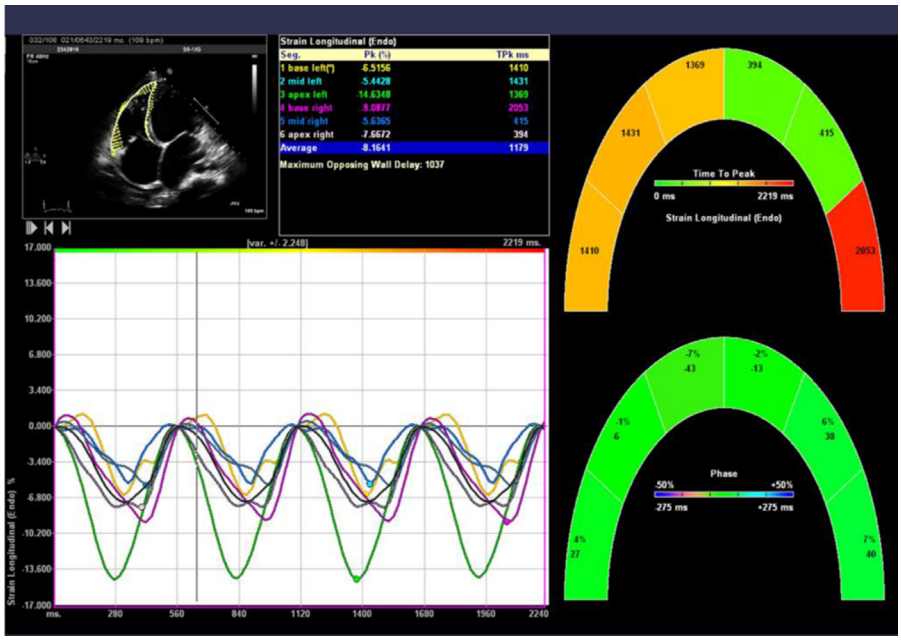

Fig. 1. Example of right ventricular (RV) longitudinal strain curves. The RV endocardial border was manually traced in the apical 4-chamber view, and the software calculated the strain and strain rate within the 6 myocardial segments of the right ventricle. The peak values of these segments were averaged to provide measurements of peak global RV longitudinal systolic strain, systolic strain rate, and diastolic strain rate. The lateral wall basal, middle, and apical segments were averaged to obtain lateral wall values.

significant variability due to differences in the angle of image acquisition, and may not consistently account for regional variations in contractility.[4] Additionally, echocardiographic imaging of LVAD patients may be technically limited because of shadowing and artifact related to the LVAD hardware itself, making consistent measurement of these indices of RV function difficult.

Myocardial strain (a dimensionless measure of tissue deformation) and strain rate (the speed at which tissue deformation occurs) as measured by speckle-tracking echocardiography are quantitative, angle-independent, reproducible, and more sensitive assessments of RV systolic and diastolic function compared with conventional echocardiography.[5] RV longitudinal strain and strain rate as measured by speckle-tracking echocardiography have been shown to correlate well with RV stroke-work index (RVSWI)[6] and may have prognostic utility in a number

Table 1. Baseline Patient Demographics, n (%)

Total no of patients	17
Age (y)	58.4 ± 15.2
Patients with NYHA IV symptoms	17 (100%)
Male sex	15 (88%)
Female sex	2 (12%)
Heart failure etiology	
Ischemic cardiomyopathy	11 (65%)
Idiopathic dilated cardiomyopathy	6 (35%)
Indication for LVAD placement	
Bridge to transplantation	5 (29%)
Destination Therapy	12 (71%)
INTERMACS profile level	
1	1 (6%)
2	6 (35%)
3	6 (35%)
4	4 (24%)
5–7	0 (0%)
Comorbidities	
Coronary artery disease	11 (65%)
Earlier coronary artery bypass surgery	6 (35%)
Diabetes mellitus	6 (35%)
Hypertension	13 (76%)
Chronic obstructive pulmonary disease	4 (24%)
Obstructive sleep apnea	5 (29%)
Baseline laboratory data (mean ± SD)	
Albumin (g/dL)	3.2 ± 0.5
Aspartate aminotransferase (U/L)	41.2 ± 42.2
Alanine aminotransferase (U/L)	29.2 ± 19.1
Creatinine (mg/dL)	1.3 ± 0.5
Hematocrit (%)	36.3 ± 5.1

NYHA, New York Heart Association functional class; LVAD, left ventricular assist device; INTERMACS, Interagency Registry for Mechanically Assisted Circulatory Support.

Table 2. Medications Before and After Left Ventricular Assist Device (LVAD) Implantation

Medication	Before*	After†
Beta-blocker	4 (24%)	8 (47%)
ACE inhibitor/ARB	10 (59%)	13 (76%)
Aldosterone antagonist	10 (59%)	11 (65%)
Diuretic	16 (94%)	5 (29%)
Vasodilator (eg, hydralazine, nitroprusside)	10 (59%)	6 (35%)
Digoxin	8 (47%)	4 (24%)
Phosphodiesterase inhibitor	1 (6%)	2 (12%)
Inotrope or Vasopressor	10 (59%)	0 (0%)

ACE, angiotensin-converting enzyme; ARB, angiotensin II receptor blocker.

*Medications noted before LVAD implantation included medications listed as being taken immediately before LVAD implantation.

†Medications included in this analysis included any medications listed within 1 month before the last echocardiographic follow-up study.