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Independent and Incremental Role of Quantitative Right Ventricular Evaluation for the Prediction of Right Ventricular Failure After Left Ventricular Assist Device Implantation

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Objectives This study sought to determine the utility of quantitation of right ventricular (RV) function in predicting RV failure in patients undergoing left ventricular assist device (LVAD) implantation.

Background Clinical evaluation alone seems insufficient for predicting RV failure, an important cause of morbidity and mortality after LVAD implantation.

Methods Clinical, hemodynamic, and echocardiographic data were collected on 117 patients undergoing LVAD implantation. Standard pre-procedural echocardiographic RV measurements were supplemented by velocity vector imaging of RV free wall longitudinal strain. RV failure was defined as the need for placement of an RV assist device, or the use of inotropic agents for >14 days. Receiver operating characteristic curves were derived, with resampling to generate valid estimates of prediction accuracy. A net reclassification index was calculated for comparison of risk scores.

Results RV failure occurred in 47 of 117 patients (40%). There was a significant difference in peak strain between patients with and without RV failure (–9.0% vs. –12.2%; $p < 0.01$). A peak strain cutoff of –9.6% predicted RV failure with 76% specificity and 68% sensitivity. In a multivariate logistic regression analysis including variables from the established Michigan RV risk score, peak strain remained an independent predictor of RV failure. RV strain was incremental to the Michigan risk score as a predictor of RV failure (area under the receiver operating characteristic curve: 0.77 vs. 0.66; $p < 0.01$). The net reclassification index with strain was +10.4%.

Conclusions Reduced RV free wall peak longitudinal strain was associated with an increased risk for RV failure among patients undergoing LVAD implantation. (J Am Coll Cardiol 2012;60:521–8) © 2012 by the American College of Cardiology Foundation

Implantation of a left ventricular assist device (LVAD) is increasingly used in the treatment of end-stage heart failure (1). Unfortunately, as LVAD placement does not augment the function of the right ventricle (RV), unanticipated RV failure remains a significant clinical problem (2), with rates varying between 5% and 44%, influenced by differing criteria and populations (3–16). RV failure is characterized by reduced end-organ function from a low-flow state and/or increased systemic venous pressures. No uniform definition of severe RV failure exists, but a common definition is the need for placement of a right ventricular assist device (RVAD) or the use of intravenous inotropes for >14 days post-operatively (5,6,12,17).

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The poor prognosis of RV failure after LVAD insertion may be improved if patients at risk are appropriately identified in advance and have biventricular mechanical support placed at the time of initial surgery (18). Unfortunately, risk scores for RV failure (3–15), including scoring systems that combine clinical, hemodynamic, and laboratory parameters (8,9,15), have not been rigorously tested in populations outside those in which they were derived. Echocardiographic parameters have been reported to provide valuable information about the risks of RV failure (9–11,13,14). Two-dimensional global strain imaging is a new echocardiographic technique for assessing systolic per-

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Abbreviations and Acronyms

AUC = area under the curve
LV = left ventricle
LVAD = left ventricular assist device
RV = right ventricle
RVAD = right ventricular assist device

formance based on the sum of regional deformation. Changes in RV strain have been shown to be valuable in predicting clinical outcomes in heart failure (19). In this study, we sought to evaluate the utility of RV strain for the prediction of RV failure after LVAD implantation.

Methods

Patient selection. We studied data from consecutive patients who underwent continuous-flow LVAD placement at the Cleveland Clinic Foundation from May 1, 2007, until April 30, 2011. These devices included the HeartMate II (Thoratec, Pleasanton, California) and the HeartWare HVAD (HeartWare, Oakville, California). Patients undergoing replacement of an existing LVAD, or with a pre-operative plan for biventricular support with a total artificial heart or RVAD, or who were supported with extracorporeal membrane oxygenation at the time of their echo were excluded. Patients were also excluded if they had no archived pre-operative transthoracic echocardiogram, or if image quality was deemed insufficient to perform analysis of RV function. The study protocol was approved by the institutional review board at the Cleveland Clinic.

Clinical data. Baseline clinical, demographic, hemodynamic, and laboratory data were gathered prospectively in the electronic record and entered into the ventricular assist device database. The electronic record was used to identify patients who underwent RVAD placement, as well as to determine mortality and duration of hospital stay. Inotrope and vasopressor use was also verified by review of the electronic medication ordering system.

The Michigan RV risk score (8) was calculated for each patient. This score assigns points based on 4 variables, with vasopressor use adding 4 points, creatinine >2.3 mg/dl adding 3 points, bilirubin >2 mg/dl adding 2.5 points, and aspartate aminotransferase >80 IU/dl adding 2 points. A higher score is associated with a greater risk for RV failure (8).

Echocardiographic assessment. Pre-operative transthoracic echocardiograms were reviewed and analyzed by a reader blinded to clinical outcomes. Standard echocardiographic measurements of the RV were made in accordance with current guidelines (20), including the maximal transverse RV end-diastolic dimension, end-systolic and end-diastolic RV areas, and fractional area change. Maximal systolic excursion of the tricuspid annulus was measured using 2-dimensional images.

Longitudinal strain was measured retrospectively using standard commercial software (Velocity Vector Imaging, Siemens AG, Erlangen, Germany). The endocardial border of the RV was traced from an apical 4-chamber view, and strain curves were generated automatically for each of 6 segments. The peak strain for the 3 segments corresponding to the RV free wall was averaged to produce a global longitudinal strain measurement (Figs. 1 and 2). Strain measurements were carried out retrospectively as a part of the study protocol, and as such were not available when

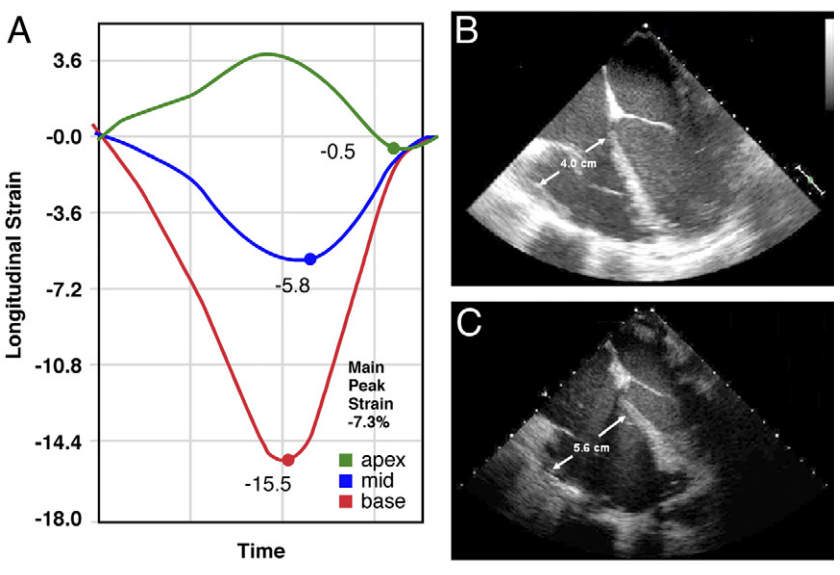

Figure 1 Pre- and Post-Operative Images in a Patient With RV Failure

Patient in whom right ventricle (RV) failure developed post-operatively and required right ventricular assist device (RVAD) implantation. **(A)** RV strain curve from pre-operative transthoracic echocardiogram showing a peak RV free wall strain of –7.3%. **(B)** RV diastolic diameter on pre-operative transesophageal echocardiography (TEE). **(C)** Increased RV diameter on TEE before RVAD implantation.

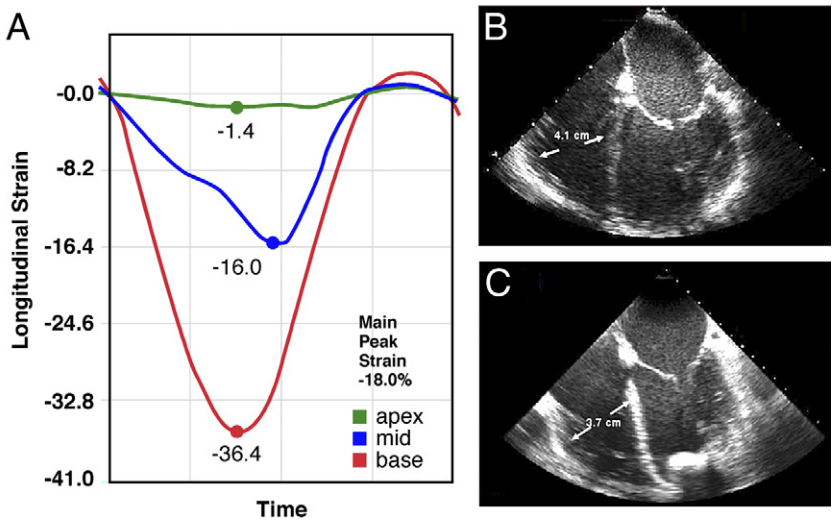

Figure 2 Pre- and Post-Operative Images in a Patient Without RV Failure

Patient in whom right ventricle (RV) failure did not develop. **(A)** RV strain curve from pre-operative transthoracic echocardiogram showing a peak RV free wall strain of –18.0%. **(B)** RV diastolic diameter on pre-operative transesophageal echocardiography (TEE). **(C)** Interval decrease in RV size on repeat TEE 28 days after left ventricular assist device implantation (performed to assess for endocarditis).

clinical decisions were made. To assess interobserver variability, global longitudinal RV strain was measured by 2 independent blinded investigators in a subgroup of 20 study patients. Intraobserver variability was measured by repeated assessment of strain in the same group of patients by 1 investigator at a separate time.

Outcomes. Patients were divided into 2 groups based on the occurrence of post-operative RV failure. RV failure was defined as unplanned insertion of an RVAD or the use of an intravenous inotrope for >14 days post-operatively.

Statistical analysis. Pre-operative variables were compared between the 2 groups using JMP Pro 9.0 (SAS Institute Inc., Cary, North Carolina). Continuous variables were compared using the unpaired *t* test for normally distributed variables and the Wilcoxon rank sum test for non-normally distributed variables. The chi-square or Fisher exact test was used for categorical variables. A p value of <0.05 was considered significant. Univariate regression analysis was performed to calculate an odds ratio for RV failure for each baseline variable.

Receiver operating characteristic curves were generated and compared (21) using MedCalc for Windows 11.6.1 (MedCalc Software, Mariakerke, Belgium). Bootstrap estimation with resampling from 1,000 simulations was used to generate valid estimates of prediction accuracy. A net reclassification index was calculated for comparison of risk scores (22).

A series of exploratory models was created by multivariate logistic regression to seek the strongest predictors of outcome, based on the greatest pseudo-R^2. Variables were entered allowing 1 variable per 10 events, and were then preferentially removed with $p > 0.05$. Candidate variables were selected on clinical grounds (including all components of the Michigan RV risk score) (8), within the categories of clinical features, hemodynamics, echo assessment of RV function, markers of end-organ function, and therapy.

Interobserver and intraobserver variability in RV strain measurement were assessed using Bland-Altman analysis for a selected group of 20 patients.

Results

Patient characteristics. Between May 1, 2007, and April 30, 2011, LVADs were implanted in 143 patients at the Cleveland Clinic who met the clinical criteria for study inclusion. Twenty-six patients were excluded on the basis of missing echocardiographic data or poor image quality, leaving a final study group of 117 patients. Two patients underwent implantation with the HeartWare HVAD (HeartWare), and the other 115 received the HeartMate II (Thoratec).

A comparison of the clinical characteristics of the 117 patients included in the study with those of the 26 patients with inadequate images for analysis is shown in Table 1. Apart from mean arterial blood pressure, excluded patients had a similar risk profile.

Outcomes. RV failure occurred in 47 of 117 patients (40%), including 10 patients who underwent RVAD placement and 37 patients who required inotropes for >14 days. The 1-year mortality rates were 9 of 47 (19%) and 13 of 70 (19%) in the groups with and without RV failure, respectively ($p = 0.94$).

Associations of RV failure. Clinical predictors of RV failure included pre-operative inotrope use, bilirubin, cardiac index, and pulmonary vascular resistance. Univariate odds ratios for RV failure of clinical and hemodynamic variables are shown in Table 2.

Table 1 Baseline Clinical, Laboratory, and Hemodynamic Characteristics of Included and Excluded Patients

Characteristic	Study Patients (n = 117)	Excluded on the Basis of Imaging (n = 26)	p Value
Age (yrs)	58 (47.5–65)	57.5 (51.5–74)	0.83
Male	92/117 (79)	23/26 (88)	0.41
Ischemic etiology	47/70 (40)	13/26 (50)	0.36
History of COPD	8/117 (7)	2/26 (8)	0.88
Previous cardiac surgery	39/117 (33)	10/26 (38)	0.62
Bridge to transplantation	78/117 (67)	18/26 (69)	0.80
Pre-operative mechanical ventilation	6/117 (5)	0/26 (0)	0.24
Pre-operative IABP/MCS	21/117 (18)	5/26 (19)	0.28
Pre-operative inotrope	72/117 (62)	18/26 (69)	0.54
Cardiopulmonary bypass time (min)	89 (69–109)	79 (60–105)	0.18
Serum sodium (mmol/l)	134 (131–137)	134 (130–137)	0.89
Blood urea nitrogen (mg/dl)	30 (22–39)	32 (22–48)	0.65
Creatinine (mg/dl)	1.27 (1.09–1.70)	1.41 (1.09–1.62)	0.53
AST (U/l)	30 (24–51.5)	27 (21.8–40.8)	0.32
Bilirubin (mg/dl)	1.1 (0.8–1.7)	1.0 (0.6–1.5)	0.08
Hemoglobin (g/dl)	11.2 (9.7–12.7)	11.2 (9.7–12.8)	0.82
Prothrombin time INR	1.1 (1.1–1.3)	1.2 (1.1–1.5)	0.12
Heart rate (beats/min)	89 (73–100)	85 (79–100)	0.99
Right atrial pressure (mm Hg)	11 (6–15)	11 (6–18)	0.97
Mean pulmonary artery pressure (mm Hg)	33 (28–41)	35 (32–40)	0.29
Pulmonary capillary wedge pressure (mm Hg)	20 (17–26)	23 (16–27)	0.69
Mean systemic arterial pressure (mm Hg)	74 (68–79)	79 (72–85)	0.04
Cardiac index (l/min)	2.25 ± 0.59	2.06 ± 0.43	0.96
Pulmonary vascular resistance (WU)	2.4 (1.5–4.0)	2.3 (1.8–2.8)	0.94
RV stroke work index (mm Hg ml/m^2 beat)	542 (367–741)	501 (390–712)	0.66

Values are median (interquartile range), n/N (%), or mean ± SD.
AST = aspartate aminotransferase; COPD = chronic obstructive pulmonary disorder; IABP = intra-aortic balloon pump; INR = international normalized ratio; MCS = mechanical circulatory support; RV = right ventricular.

Table 3 outlines the association of pre-operative echocardiographic parameters with RV failure. RV failure was associated with subjective assessment of RV function as moderate to severely reduced (51% vs. 30%; p = 0.04) and with lower peak longitudinal strain of the RV free wall (−9.0% [interquartile range: −7.3% to 11.4%] vs. −12.2% [interquartile range: −9.5% to −14.9%]; p < 0.01).

Using bootstrapping with 1,000 simulations, a receiver operating characteristic curve (area under the curve [AUC]: = 0.70) was used to select an RV strain cutoff of −9.6% (specificity: 76%; sensitivity: 68%) to predict RV failure.

Independent associations of RV failure. A number of predictive models were created to seek the strongest predictors of RV failure. Variables were entered into the model 4 at a time (allowing 1 variable per 10 events). When combined with the Michigan RV risk score in a multivariate model, RV peak longitudinal strain was a significant contributor to the model (Table 4). It remained a significant variable in all tested models.

Incremental prediction of RV failure. We then set out to quantify the added value of RV strain when combined with the Michigan risk score. In this analysis, an RV peak longitudinal strain of >−9.6% was assigned a weighting of 2.5 points based on the relative odds ratios for RV failure of creatinine and bilirubin in our cohort. The Michigan RV risk score was calculated for each patient and compared with this modified risk score. Receiver operating characteristic curves for the Michigan risk score, and the score combined with RV strain, are shown in Figure 3. A similar score was calculated by assigning a weighting of 2.5 points to subjective echo assessment of RV dysfunction (Fig. 3). There was a significant difference between the AUC for the Michigan risk score and the combined score with RV strain (AUC: 0.66 vs. 0.77; p < 0.01). There was no significant difference between the AUC for the Michigan risk score and that of a combined score using subjective assessment of RV function (AUC: 0.69; p = 0.33). Using a cutoff of ≥3 for the new score incorporating RV strain, 8 of 45 patients with RV failure would be correctly reclassified as "at risk," and 5 of 68 patients without RV failure would be incorrectly reclassified as at risk, leading to a net reclassification index of +10.4%.

Reliability of RV strain. When peak longitudinal strain was measured in the same 20 patients by 2 blinded observers, there was a bias of 0.8% and SD of 2.9%. Intraobserver comparisons yielded a bias of 0.1% and a SD of 2.8%.

Discussion

The findings of this study indicate that RV strain may be a useful pre-operative predictor of RV failure in patients

Table 2 Clinical, Laboratory, and Hemodynamic Risk Factor for RV Failure

Characteristic	OR for RV Failure	95% Confidence Interval	p Value
Age	0.98	0.95–1.01	0.13
Male	0.54	0.22–1.32	0.17
Ischemic etiology	1.02	0.48–2.17	0.96
History of COPD	0.47	0.12–1.87	0.36
Previous cardiac surgery	0.65	0.29–1.45	0.32
Bridge to transplantation	0.59	0.27–1.29	0.23
Pre-operative mechanical ventilation	3.16	0.56–18.20	0.22
Pre-operative IABP/MCS	1.45	0.56–3.75	0.45
Pre-operative inotrope	2.53	1.13–5.68	0.03
Cardiopulmonary bypass time	1.00	0.99–1.01	0.69
Michigan RV risk score (per point)	1.48	1.17–1.91	<0.01
Pre-operative vasopressor	2.08	0.44–9.73	0.35
Creatinine	1.42	0.66–3.10	0.36
AST	1.00	1.00–1.01	0.57
Bilirubin	1.68	1.17–2.62	<0.01
Serum sodium	0.95	0.88–1.02	0.17
Blood urea nitrogen	1.01	0.98–1.03	0.62
Hemoglobin	1.14	0.92–1.41	0.23
Prothrombin time INR	3.12	0.75–22.2	0.12
Heart rate (per 10 beats/min)	1.16	0.93–1.44	0.17
Right atrial pressure	1.00	0.94–1.06	0.94
Mean pulmonary artery pressure	0.99	0.94–1.04	0.61
Pulmonary capillary wedge pressure	0.96	0.89–1.02	0.20
Mean systemic arterial pressure	0.97	0.92–1.02	0.30
Cardiac index	0.41	0.19–0.82	0.01
Pulmonary vascular resistance	1.45	1.13–1.91	<0.01
RV stroke work index (per 100 mm Hg ml/m^2 beat)	0.88	0.77–1.01	0.05

OR = odds ratio; other abbreviations as in Table 1.

undergoing LVAD implantation—more powerful than any other echocardiographic parameter studied, including tricuspid annular systolic excursion (14,23) and RV-to-LV diameter ratio (11). Global longitudinal RV strain was an independent predictor of RV failure, which was incremental to a currently used risk model (8,9), correctly reclassifying a significant number of patients.

RV function in LVAD recipients. After LVAD implantation, adequate RV function is required to permit antegrade inflow into the device. RV failure occurs when the RV provides inadequate output, or when it provides output only at the expense of high filling pressures. Unfortunately, many patients undergoing LVAD implantation have some degree of RV dysfunction prior to surgery, which can put them at risk for RV failure. In many patients, RV function improves after implantation, probably because RV afterload is reduced by reversal of pulmonary venous hypertension (16,24). However, complex hemodynamic changes at the time of LVAD implantation can adversely affect RV function (25). Increased forward output from the left heart and LV decompression cause a leftward shift of the interventricular septum, which may interfere with normal RV mechanics. Post-operative RV distension and increased filling pressures may result from perioperative use of blood products and crystalloid, and RV dysfunction may be exacerbated by intraoperative RV injury (due to poor cardioprotection, right coronary air embolus, and/or pulmonary hypertension related to cardiopulmonary bypass [26]).

RV failure has been associated with adverse outcomes, including mortality. However, the financial cost associated with implanting an RVAD in all patients makes routine biventricular support an unacceptable option. Moreover, biventricular support with both an LVAD and an RVAD may lead to a greater number of device-related complications. The majority of RVADs being used for long-term support are early generation pumps using pulsatile pneumatic drive systems. They tend to have larger control systems and limited portability and carry a greater risk for mechanical failure and hemolysis (27). These more cumbersome devices impose greater complexity to long-term care and may reduce quality of life. Thus, although intervening prophylactically by placing biventricular support at the time of initial surgery is more effective than responding with a "rescue" RVAD (18,26), a selective approach to RVAD implantation is more desirable than widespread use.

Assessment of RV function. The adequacy of RV function (and its capacity to respond to a change in loading conditions after LVAD implantation) is difficult to evaluate. Previous work has highlighted the relevance of markers of end-organ function and congestion, such as urea, creatinine,

Table 3 Echocardiographic Risk Factor for RV Failure

Echocardiographic Variable	No RV Failure	RV Failure	OR (95% CI)	p Value
LV end-diastolic dimension (cm)	7.0 ± 1.0	6.9 ± 1.1	0.86 (0.58–1.27)	0.45
LV ejection fraction (%)*	15 (10 to 20)	15 (10 to 25)	1.01 (0.93–1.11)	0.75
Mitral regurgitation (moderate to severe)	33/62 (53)	24/44 (55)	1.03 (0.70–1.52)	0.89
Subjective RV dysfunction (moderate to severe)	19/63 (30)	22/43 (51)	1.56 (1.04–2.34)	0.03
Tricuspid regurgitation (moderate to severe)	21/61 (34)	16/44 (36)	1.04 (0.69–1.56)	0.84
Lateral RV peak longitudinal strain (%)	−12.2 (−9.5 to 14.9)	−9.0 (−7.3 to 11.4)	0.84 (0.75–0.92)	<0.01
RV fractional area change (%)	22.1 ± 8.4	19.4 ± 8.7	0.96 (0.92–1.01)	0.09
RVEDD-to-LVEDD ratio	0.72 ± 0.15	0.74 ± 0.17	2.75 (0.24–33.8)	0.42
Tricuspid annular systolic excursion (cm)	1.32 ± 0.29	1.22 ± 0.25	0.26 (0.06–1.05)	0.06

Values are mean ± SD, median (interquartile range), or n/N (%).
LV = left ventricular; LVEDD = left ventricular end-diastolic dimension; RVEDD = right ventricular end-diastolic dimension; other abbreviations as in Tables 1 and 2.

Table 4 Predictive Model for RV Failure, Including Michigan Risk Score and Selected Echocardiographic Variables

Variable	SD	OR (95% CI) per SD	OR (95% CI)	p Value
Michigan Risk score	1.8	2.01 (1.30–3.28)	1.48 (1.16–1.95) (per 1-point increase)	<0.01
Lateral RV peak longitudinal strain, %	4.3	0.53 (0.31–0.85)	0.86 (0.76–0.96) (per 1% increase)	0.01
Tricuspid annular systolic excursion, mm	2.8	0.82 (0.50–1.33)	0.93 (0.78–1.11) (per 1-mm increase)	0.43
RV-to-LV diameter ratio	0.16	1.03 (0.68–1.57)	1.02 (0.78–1.34) (per increase of 0.1)	0.89

CI = confidence interval; LV = left ventricle; OR = odds ratio; RV = right ventricle.

aminotransferases, bilirubin, and prothrombin time. Unfortunately, these markers are not specific to RV dysfunction, and they may be insensitive as they reflect only the degree of pre-existing baseline RV failure. Nonetheless, such readily available parameters continue to be of value, and in our patients, bilirubin was a strong independent predictor of RV failure (Table 2).

Pre-operative hemodynamic measures have had variable predictive power in different studies. In the present work, cardiac index and pulmonary vascular resistance were both related to the rate of RV failure. The lower cardiac index in the patients in whom RV failure developed may have reflected a group of patients in whom baseline RV dysfunction was sufficient to limit cardiac output. Those with greater pulmonary vascular resistance might be less likely to have an improvement in forward flow when the LV is decompressed. RV stroke work index and right atrial or central venous pressure, which have previously been found to be helpful (3,7,12), were not significant predictors in our group perhaps, in part, because these variables are used at our institution to delay LVAD surgery until management can be altered, and to select patients for planned biventricular support.

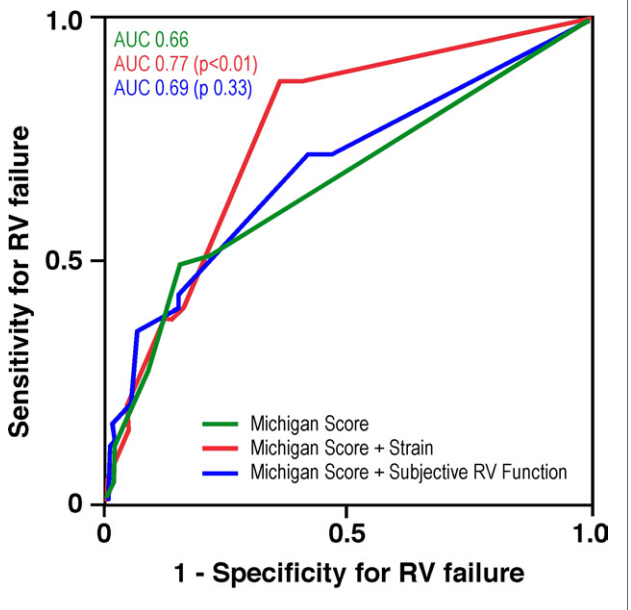

Figure 3 Michigan Risk Score With the Addition of Echocardiographic Assessment of RV Function by Subjective Reporting and RV Strain

Displayed p values are for the comparison of AUC to the Michigan risk score alone. AUC = area under the receiver operating characteristic curve; RV = right ventricle.

RV imaging is an attractive adjunct to clinical RV evaluation because it is noninvasive and may offer greater sensitivity to change than markers of pre-existing RV failure. However, it is limited by the complex geometry of the RV (20,28). RV size (assessed as RV-to-LV diameter ratio) has previously been reported as a useful finding on transesophageal echocardiography (6,11), but it was not discriminatory in this group. Measures of RV systolic performance range from subjective evaluation to fractional area change and tricuspid annular systolic excursion. Tricuspid excursion has been previously reported to have good predictive value for RV failure (14), although such displacement parameters may be influenced by the rocking motion of the heart that may occur with severe LV dysfunction. Longitudinal strain provides a direct measure of regional deformation and may help to delineate more subtle abnormalities of RV contractility than other echo variables, such as subjective RV function or fractional area change. Strain is likely a composite measure of RV dysfunction and loading, as decreased global RV strain is seen in patients with pulmonary hypertension (23).

Study limitations. Significant differences in RV strain were seen between those with and without RV failure. On its own, however, the predictive value of this variable was only modest, with a sensitivity of 68% and a specificity of 76%. These findings highlight the continued importance of incorporating multiple factors into any method of risk assessment. The number of events in our population limited the number of parameters that could be incorporated into our process of multivariate modeling. For this reason, we focused on the analysis of RV strain as an adjunct to the existing Michigan RV risk score.

Feasibility is an important limitation of the approach described here for LVAD risk prediction. In this study, 26 of 143 clinically eligible patients (18%) were excluded because of insufficient or unavailable images, in most cases

(n = 23 [16%]) because of poor image quality. Further experience with the method may improve feasibility—especially with obtaining dedicated views of the RV. However, this technique will likely remain unsuitable for patients with poor acoustic windows or very large RVs. Measurements of strain are difficult in the thin-walled RV, which may contribute to the detection of only fair interobserver agreement using this technique. The relative merits of different strain measurement approaches are undefined; it is likely that different RV strain measures would be obtained using different software (29).

RV failure after LVAD implantation can be defined in a variety of ways, and criteria have varied considerably between reports. We selected a commonly employed definition that incorporates the perceived clinical significance of severe RV dysfunction, but with the limitation of some degree of subjectivity. The use of pulmonary vasodilators such as nitric oxide was not assessed, which might have led to an underestimation of the number of patients who required intensive treatment for RV dysfunction. However, in our study, the frequency of RV failure was in the upper range of what has been reported in the literature. This finding may have been an artefact of local policies regarding aggressive treatment with inotropes and early intervention with "rescue" RVADs to prevent the deleterious effects of RV failure. Less aggressive treatment at other centers might lead to a lower incidence of RV failure. On the other hand, aggressive treatment may have helped to mitigate some of the risk associated with RV failure, perhaps contributing to the lack of difference in mortality between patients with and without RV failure in our study.

It should be acknowledged that this study was carried out at a single institution. As such, the reproducibility of these findings in other populations remains untested. The necessary exclusions from this study may also limit generalizability. Because RV strain is a load-dependent measure, patients who were being supported with extracorporeal membrane oxygenation at the time of imaging were excluded from this study, and the results cannot be extrapolated to this group. We also excluded patients undergoing pulsatile LVAD implantation in order to reflect contemporary device therapy. The overwhelming majority of our patients received the HeartMate II LVAD. Although neither of the patients who received the HeartWare LVAD experienced RV failure, the numbers are too small to allow for extrapolation about the differences between RV failure rates of the 2 devices.

This study tested the use of RV strain as a part of a pre-operative strategy for risk assessment. The utility of this measurement in combination with other parameters as part of an intraoperative strategy using transesophageal echocardiography has not been explored. Furthermore, the baseline echocardiograms were obtained only under a single set of hemodynamic conditions, which varied between patients. It is possible that the response of RV strain to changes in preload, afterload, or contractility may provide additional prognostic information.

Conclusions

The findings from this study support the findings of previous reports that RV failure is a common post-operative issue after LVAD insertion. Global longitudinal strain of the RV free wall represents a new parameter that may help to predict the occurrence of this serious outcome in patients receiving LVADs. It may be of value in making clinical decisions about device selection for such patients in the future, either in combination with other clinical factors, such as those found in the Michigan risk score, or in other risk-scoring systems.

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