Published in final edited form as:

J Heart Lung Transplant. 2013 June ; 32(6): 603–608. doi:10.1016/j.healun.2013.03.004.

Right ventricular stroke work index as a negative predictor of mortality and initial hospital stay after lung transplantation

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Abstract

BACKGROUND—Studies have shown that patients with poor pre-lung transplant (LTx) right ventricular (RV) function have prolonged post-operative ventilation time and intensive care stay as well as a higher risk of in-hospital death. RV stroke work index (RVSWI) calculates RV workload and contractility. We hypothesized that patients with higher RV workload capacity, indicated by higher RVSWI, would have better outcomes after LTx.

METHODS—A retrospective record review was performed on all LTx patients between 2005 and 2011 who had right heart catheterizations (RHC) 1-year before LTx. In addition, results for echocardiograms and cardiopulmonary exercise testing within 1-year of RHCs were gathered.

RESULTS—Mean RVSWI was 9.36 ± 3.59 for 115 patients. There was a significant relation between mean pulmonary artery pressure (mPAP), RVSWI, RV end-diastolic diameter (RVEDd), left atrial dimension (LAD), peak and resting pressure of end-tidal carbon dioxide, minute ventilation /volume of carbon dioxide production, and 1-year mortality after LTx. Contrary to our hypothesis, those who survived had lower RVSWI than those who died within 1 year (8.99 ± 3.38 vs 11.6 ± 4.1, $p = 0.026$). Hospital length of stay significantly correlated with mPAP, RVSWI, left ventricular ejection fraction, percentage of fractional shortening, RVEDd, RV fractional area change, LAD, and RV wall thickness in diastole. Intensive care length of stay also significantly correlated with these variables and with body mass index. RVSWI was significantly different between groups of different RV function, indicating that increased RVSWI is associated with impairment of RV structure and function in patients undergoing LTx evaluation.

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Disclosure statement

None of the authors has a financial relationship with a commercial entity that has an interest in the subject of the presented manuscript or other conflicts of interest to disclose.

CONCLUSIONS—This study demonstrates an association between 1-year mortality, initial hospital and intensive care length of stay, and pre-LTx RVSWI. Increased mPAP is a known risk for outcomes in LTx patients. Our findings support this fact and also show increased mortality with elevation of RVSWI, demonstrating the value of RV function in the assessment of risk for pre-LTx patients.

Keywords

lung transplant; right ventricular stroke work index; right heart catheterization; right ventricular function; cardiopulmonary exercise test

Lung transplantation (LTx) has been a viable treatment for end-stage lung disease for almost 30 years. However, due to donor shortages, factors predicting benefit from the procedure and clinical outcomes after LTx are warranted. It is important to risk stratify the use of grafts so that the selection of patients who receive a transplant represents those with the most realistic prospects of positive long-term outcomes.[1] Because 2 of the main risk factors for cardiovascular disease—smoking and increased age—are frequently found in LTx recipients, centers perform intensive pre-transplantation cardiovascular evaluations, including echocardiography for assessment of right ventricular (RV) structure and function and invasive right heart catheterization (RHC) before listing patients.[2] Additionally, prior studies have shown that patients with poor pre-operative RV function have prolonged post-operative ventilation time, longer intensive care stay, and are at higher risk of in-hospital death.[3]

The RV stroke work index (RVSWI) calculates RV workload and contractility based on invasive hemodynamics and patient characteristics. Most frequently, RVSWI is used to predict RV failure after left ventricular assist device insertion for advanced left ventricular failure.[4,5] Because RVSWI incorporates ventricular function and hemodynamics, we felt this would possibly be a sensitive variable to assist in the clinical management of patients considered for LTx. We hypothesized that patients with higher RV workload capacity, indicated by a higher RVSWI, would have better outcomes after LTx. To assess this hypothesis, we aimed to (1) evaluate RVSWI measured within 1 year before LTx and outcomes after LTx, including survival, intubation time, hospital and intensive care unit length of stay, and (2) compare RVSWI vs pulmonary hypertension (PH), and exercise and echocardiographic variables, and their association to mortality and length of stay.

Methods

The New York Presbyterian-Columbia University Medical Center Institutional Review Board approved this study.

Study cohort

A retrospective record review was performed of all patients who received LTx between 2005 and 2011, after the implementation of the new Lung Allocation Score (LAS) at New York Presbyterian-Columbia University Medical Center, with RHCs performed at our center within 1-year before LTx. Additionally, echocardiograms and cardiopulmonary exercise testing (CPET) results within 1 year of RHC were gathered when available. Parameters

analyzed from echocardiograms included the left ventricular (LV) end-diastolic and end-systolic internal dimension, interventricular septal wall thickness in diastole, posterior wall thickness in diastole, LV ejection fraction, LV percentage of fractional shortening, left atrial dimension (LAD), right ventricular (RV) end-diastolic diameter (RVEDd), RV fractional area change (RVFAC), and RV wall thickness in diastole in accordance with the recommendations of the American Society of Echocardiography. RVFAC was obtained by tracing the RV endocardium in systole and diastole from the annulus, along the free wall to the apex, and then back to the annulus, along the interventricular septum.[6] The qualitative assessments of RV function were obtained from the institutional echocardiogram reports, which were classified into normal, mildly reduced, moderately reduced, and severely reduced RV function.

Variables from CPET included minute ventilation (V_E), oxygen uptake (V_{O_2}), rate of carbon dioxide production (V_{CO_2}), systolic and diastolic blood pressure, heart rate, pressure of end-tidal carbon dioxide (Pet_{CO_2}), and oxygen saturation. Basic metabolic and hepatobiliary laboratory results were reviewed if they were within 6 months of RHC. These included blood urea nitrogen, creatinine, total protein, albumin, total bilirubin, direct bilirubin, amino alanine transaminase, and total alkaline phosphatase. Death within 1 year of LTx was determined from the Social Security database. Hospital length of stay after LTx, intensive care unit length of stay, and endotracheal intubation time were all gathered from patients' records.

Invasive hemodynamics using RHC

Catheterization was performed at rest with a Swan-Ganz catheter. Variables collected included the mean right arterial pressure (mRAP), mean pulmonary capillary wedge pressure, mean pulmonary artery pressure (mPAP), heart rate, cardiac output, and cardiac index when available. Stroke volume index (SVI) was calculated by the cardiac index/heart rate × 1,000. PH was defined as mPAP ≥ 25 mm Hg at rest.[7,8] RVSWI was calculated by the following equation[9]: RVSWI = SVI × (mPAP − mRAP) × 0.0136

Statistical analyses

Data analyses were completed using SPSS 19 software (IBM Inc, Armonk, NY). Continuous data were evaluated for normality using the Kolmogorov-Smirnov test. Normally distributed data are presented as mean ± standard deviation and non-normal data as median and interquartile range (IQR; 25%–75%). Group differences between survivors and non-survivors were established with the use of 2-sided *t*-tests for independent samples for continuous variables and chi-square tests for categoric variables. Analyses of group differences for non-normally distributed variables were performed with the Mann-Whitney *U* test. One-way analysis of variance was used to determine if RV function was associated with RVSWI. Correlations between RVSWI and other variables were assessed using Pearson's correlations. To evaluate the capacity of variables to predict the risk of death within 1 year of LTx, Cox proportional hazards regression analyses were used.[10] Vital status was censored on June 13, 2011. Lastly, to compare the precision of RVSWI vs mPAP alone, we computed the C statistics for a model containing mPAP or RVSWI as the sole independent variable for detecting death within 1 year of LTx. Statistical significance was

set a priori at an $\alpha = 0.05$ and 95% confidence intervals (CI) were determined for hazard ratios (HR).

Results

Of the 230 LTxs were performed between January 2005 and March 2011, RHCs were performed in 135 (57%) within 1 year before LTx (164 ± 96 days; median, 155 days [IQR, 78–229]). This cohort was 39% female with an average age of 52 ± 14 years. The mean RVSWI was 9.36 ± 3.59 for the 115 patients who had complete RHCs. The diagnoses were 65 patients with interstitial lung disease, 27 with cystic fibrosis, 21 with chronic obstructive pulmonary disease, 10 with sarcoidosis, 6 re-LTxs, 3 with bronchiectasis, 2 with pulmonary arterial hypertension, and 1 with eosinophilic granuloma. We collected information on endothelin receptor antagonists, calcium-channel blockers, diuretics, cyclic guanosine monophosphate (cGMP)-specific phosphodiesterase inhibitors, and dobutamine. Ninety-four of the patients were not taking any of these medications close to the time of their RHC, 7 were taking endothelin receptor antagonists, 16 were taking calcium-channel blockers, 29 were taking diuretics, 4 were taking cGMP-specific phosphodiesterase inhibitors, and 1 was taking dobutamine. There were no differences in medications between those who survived and died within 1 year of LTx, determined by a chi-square test (data not shown).

Relation with 1-year mortality

Comparisons of those who survived and died within 1-year from LTx for each of the pre-operative recipient demographic variables and associated HRs for univariate predictors of 1-year mortality with a $p \leq 0.05$ are reported in Table 1. Diagnosis was not a predictor of 1-year mortality in Cox regression (data not shown). The time to death for the non-survivors was 127 ± 107 days, whereas the follow-up time for the survivors was 1073 ± 598 days ($p < 0.001$). There were no differences in time between RHC and LTx between the 2 groups (data not shown). The causes of death were pneumonia in 10, primary graft dysfunction in 4, and in 1 patient each, cardiac arrest, lung cancer in the native lung, multiorgan failure, and peri-operative death. Those who died of primary graft dysfunction had a higher RVSWI than those who died of other causes: 13.99 (IQR, 10.11–17.86) vs 7.87 (IQR, 6.89–10.71; $p = 0.029$).

The LAS at time of transplant was 66 (IQR, 47–78) for the mortality group and 46 (IQR, 38–62) in the 112 survivors that had LAS available ($p = 0.030$). The LAS had a HR of 1.024 (95% CI, 1.002–1.046; $p = 0.035$).

The basic metabolic and hepatobiliary laboratory values were not hazardous (data not shown). Additionally, unilateral or bilateral or a left or right LTx was not significantly hazardous (data not shown).

The univariate Cox regression analysis showed a significant relation between mPAP, RVSWI, RVEDd, LAD, peak and resting Pet_{CO_2}, peak V_E/V_{CO_2}, and 1-year mortality after LTx. Those who survived had a significantly lower RVSWI than those who died within 1-year of LTx (8.99 ± 3.38 vs 11.6 ± 4.1, $p = 0.026$). The univariate Cox regression analysis also showed that for each unit increase in RVSWI, the HR for death within 1 year was 1.177

(95% CI, 1.045–1.325), whereas the HR for mPAP was 1.033 (95% CI, 1.000–1.066). The C statistic for the ability of RVSWI to predict the risk of 1-year mortality after LTx was 0.681 compared with 0.655 with the use of mPAP alone. The LAS had a lower C statistic of 0.660, which was lower than the RVSWI C statistic.

Relation with hospital stay

The hospital length of stay after LTx was significantly correlated with mPAP, RVSWI, LV ejection fraction, LV percentage of fractional shortening, RVEDd, RVFAC, LAD, RVWTd, and LAS (Table 2). Length of stay in the intensive care unit was also significantly correlated with these variables and with body mass index ($r = 0.218$, $p = 0.027$). No variables correlated with intubation time (data not shown).

Variables associated with RVSWI

The LAS, CPET variables, and RV function measures on echocardiogram that were significantly associated with RVSWI are reported in Table 3. One-way analysis of variance determined that RVSWI was significantly different between groups of RV function, indicating that increased RVSWI is associated with an impairment of RV structure and function in patients undergoing LTx evaluation (Table 4).

Discussion

Our current study demonstrates an association between increased 1-year mortality, length of initial hospital and intensive care unit stay, and pre-LTx RVSWI. Improving the prediction of outcomes for LTx recipients before LTx and determining risk factors for poor outcome after LTx are important because they may have an effect on the evaluation process of transplant candidates and the treatment process during time on the waiting list.[3]

Because RVSWI is a measure of RV workload, we hypothesized that patients with a lower RVSWI would have worse outcomes after transplant, corresponding to a decreased RV function before transplant. Contrary to our hypothesis of a decreased RVSWI being hazardous, the results showed that an increased RVSWI was associated with increased risk of 1-year mortality after LTx. Mean PAP is an important factor contributing to increased risk associated with RVSWI in this population. This is in agreement with the known hazards of increased pulmonary pressures in end-stage lung disease and may account for part of the increased association between RVSWI and death. The correlation of poorer outcome with higher RVSWI is a marker of the pernicious effects of elevated PA pressures. This can be seen through the increased RVSWI in patients who died of primary graft disfunction compared with other causes. Further evidence for the role of PH in the prediction of outcomes is that decreased Pet_{CO_2} and increased V_E/V_{CO_2}, which are exercise-derived markers of PH,[11,12] were also significantly correlated with RVSWI.

The echocardiographic data also support the key role of preserved RV function in the face of elevated mPAP in improved survival. RVFAC, an assessment of RV systolic function, was negatively correlated with RVSWI, and RVEDd, a measure of RV overload, was positively correlated with RVSWI. In addition, the RVWT was increased, showing a possible

compensatory RV hypertrophy in addition to the mild dilation due to the increased pulmonary pressure.

Because increased mPAP is known to be a mortality risk in end-stage lung disease, under Policy 3.7.6.4- Lung Candidates with Exceptional Cases, LTx candidates diagnosed with PH, who are deteriorating on optimal therapy and have a RAP > 15 mm HG or a cardiac index < 1.8 liters/min/m^2, can qualify for an increase in their LAS.[13] Pre-operative PH also increases mortality after heart, kidney, and liver transplantation.[3,14–17] In acute respiratory failure, where PH and increased pulmonary vascular resistance are part of regulation of pulmonary blood flow, elevation of PAP results in increased RVSWI at a given stroke volume. The increased RVSWI correlates with a more uniform ratio of ventilation to perfusion.[18,19] Similar mechanisms may play a role in pre-transplant patients, with chronically elevated mPAP eventually leading to RV failure and a decrease in RVSWI.

We believed that the chronic nature of the PH would cause a decrease in RVSWI in sicker patients that led us to our initial hypothesis. However, we found that the patients seemed for the most part to undergo transplantation in the earlier part of this trajectory, with RVSWI still intact despite the presence of PH. Thus our findings support the importance of including PH in the LAS, thereby allowing sicker patients to receive an allograft first and achieving the goal of Zalunardo et al.[3] However, our finding that higher RVSWI is associated with increased 1-year mortality indicates that further work needs to be done to assess the role of RV failure in the setting of PH on outcomes after transplant.

Another feature of our data is that the comparison of RVSWI vs mPAP alone shows that RVSWI has a slightly higher C statistic and a higher hazard ratio for detecting 1-year mortality after LTx. RVSWI also was better correlated with length of hospital and intensive care unit stay than mPAP, suggesting that the additional contribution of RV function to the calculation of RVSWI may still have a significant role in determining outcomes after transplant. Although the mPAP is only a measure of pulmonary pressures, RVSWI incorporates an assessment of RV stroke volume into an overall measure of RV workload capacity. The additional effect of RV function from RVSWI may deserve further assessment and may indicate a need to consider RV stroke volume as well as mPAP in risk stratification of patients with end stage lung disease.

In conclusion, increased mPAP is a known risk for outcomes in patients who undergo LTx. Our findings support this idea and also show an increased mortality with elevation of RVSWI, potentially demonstrating the value of RV function in the assessment of risk for pre-transplant patients. This intriguing finding suggests that future research is also needed to look at the role that RV function may play, in addition to pulmonary pressures, in acute decompensation in patients with severe lung disease.

Acknowledgments

This work was in part funded by the VIDDA Foundation.

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Table 1

Baseline Clinical Demographics and Hazard of 1-Year Mortality After Lung Transplantation

Variables[a]	Survivors (*n* = 117)	Non-survivors (*n* = 18)	HR (95% CI)[b]	*p*-value[c]
Demographics				
Age, years	56 (41–62)	59 (52–64)		0.245
Sex.				0.284
Male	69	13		
Female	48	5		
Body mass index, kg/m^2	25.3 ± 4.7	25.7 ± 4.9		0.758
Hemodynamic variables				
Mean PAP, mm Hg	25 (20–30)	30 (23–41)	1.033 (1.000–1.066)	0.035
Mean RAP, mm Hg	5.0 ± 4.5	4.8 ± 4.6		0.863
Stroke volume index, ml/m^2/beat	31.3 ± 8.7	32.6 ± 10.5		0.656
RVSWI, g/m^2/beat	8.99 ± 3.38	11.6 ± 4.1	1.177 (1.045–1.325)	0.026
Echocardiograph variables				
LVEDd, mm	42 (40–46)	43 (42–46)		0.518
LVESd, mm	28.3 ± 4.5	28.9 ± 3.3		0.510
IVSTD, mm	10 (9–11)	11 (9–12)		0.073
PWTD, mm	10 (9–11)	10 (9–12)		0.287
LVEF, %	58 ± 9	57 ± 9		0.569
%FS	34.5 ± 6.5	33.5 ± 6.3		0.554
RVEDd, mm	36 (32–39)	39 (34–43)	1.132 (1.020–1.256)	0.018
RVFAC, %	40.6 ± 7.8	37.2 ± 9.0		0.180
LAD, mm	34 (32–36)	38 (35–40)	1.092 (1.001–1.192)	0.008
RVWTd, mm	9 (8–11)	10 (8–11)		0.197
Estimated PA	42 (37–55)	49 (42–64)		0.053
Mean PAP, mm Hg	35.5 ± 10.9	41.1 ± 11.2		0.083
CPET variables				
Peak V_E, l/min	37 ± 17	44 ± 19		0.219
Peak V_{O_2}, ml/kg/min	13.56 ± 4.81	12.00 ± 3.11		0.439
V_{O_2} % predicted	41 ± 19	39 ± 17		0.665
Peak V_{CO_2}, ml/kg/min	13.49 ± 5.28	12.68 ± 4.56		0.791
Peak BP, mm Hg				
Systolic	160 ± 20	160 ± 23		0.998
Diastolic	82 ± 11	81 ± 10		0.806
Heart rate, beats/min				
Resting	97 ± 16	95 ± 15		0.744
Peak	122 ± 17	120 ± 21		0.702
Pet_{CO_2}, mm Hg				
Peak	39.9 ± 11.7	31.7 ± 8.5	0.939 (0.893–0.987)	0.003
Resting	37.0 ± 8.3	31.9 ± 5.2	0.920 (0.856–0.988)	0.003

Variables[a]	**Survivors (*n* = 117)**	**Non-survivors (*n* = 18)**	**HR (95% CI)**[b]	***p*-value**[c]
Peak V_E/V_{CO_2},	39 ± 12	46 ± 10	1.033 (1.000–1.067)	0.022

%FS, percentage of fractional shortening; BP, blood pressure; CPET, Cardiopulmonary exercise testing; IVSTD, interventricular septum wall thickness in diastole; LAD, left atrial dimension; LVEDd, left ventricular end-diastolic internal dimension; LVEF, left ventricular ejection fraction; LVESd, left ventricular end-systolic internal dimension; PAP, pulmonary arterial pressure; PetCO2, pressure of end-tidal carbon dioxide; PWTD, posterior wall thickness in diastole; RAP, right atrial pressure; RVEDd, right ventricular end-diastolic diameter; RVFAC, right ventricular fractional area change; RVSWI, right ventricular stroke work index; RVWTD, right ventricular wall thickness in diastole; VCO2, carbon dioxide production; VE, minute ventilation; VO2, volume of oxygen consumed.

[a] Continuous data are shown as mean ± standard deviation or median (interquartile range) and categoric data as number.

[b] Hazard ratios (HR) and 95% confidence intervals (CI) are shown for univariable predictors with $p \leq 0.05$.

[c] *p*-Value for between-group comparisons.

Table 2

Pearson's Correlations for Clinical Outcomes Associated with Right Ventricular Stroke Work Index

Variable	Length of stay			
	Hospital		ICU	
	r	*p*-value	*R*	*p*-value
mPAP	0.177	0.040	0.177	0.046
RVSWI	0.199	0.033	0.195	0.041
LVEF	−0.258	0.005	−0.285	0.002
%FS	−0.227	0.014	−0.247	0.009
RVEDd	0.193	0.037	0.226	0.017
RVFAC	−0.272	0.003	−0.267	0.004
LAD	0.197	0.033	0.230	0.015
RVWTd	0.215	0.020	0.212	0.025
LAS	0.203	0.021	0.197	0.029

%FS, left ventricular percentage of fractional shortening; ICU, intensive care unit; LAD, left atrial dimension; LAS, Lung Allocation Score; LVEF, left ventricular ejection fraction; MPAP, mean pulmonary arterial pressure; RVEDd, right ventricular end-diastolic diameter; RVFAC, right ventricular fractional area change; RVSWI, right ventricular stroke work index; RVWTD, right ventricular wall thickness in diastole.

Table 3

Pearson's Correlations for Variables Associated With Right Ventricular Stroke Work Index

Variable	RVSWI	
	r	p-value
Lung allocation score	0.251	0.007
CPET variables		
Peak Pet_{CO_2}	−0.348	0.001
V_E/V_{CO_2}	0.275	0.008
Peak Sp_{O_2}	−0.259	0.013
Echocardiograph variables		
IVSTD	0.319	0.001
RVEDd	0.235	0.019
RVFAC	−0.248	0.013
Estimated PA	0.382	<0.001
RVWTD	0.324	0.001
Mean PAP	0.414	<0.001

CPET, cardiopulmonary exercise testing; Estimated PA, estimated pulmonary artery pressure; IVSTD, interventricular septum wall thickness in diastole; PAP, pulmonary arterial pressure; Pet_{CO_2}, pressure of end-tidal carbon dioxide; RVEDd, right ventricular end-diastolic diameter; RVFAC, right ventricular fractional area change; RVSWI, right ventricular stroke work index; RVWTD, right ventricular wall thickness in diastole; Sp_{O_2}, oxygen saturation; V_{CO_2}, rate of carbon dioxide production; V_E, minute ventilation.

Table 4

Right Ventricular Function Is Associated With Right Ventricular Stroke Work Index As Determined by One-Way Analysis of Variance

RV Function	No.	Mean RVSWI	p-value for ANOVA
Normal	43	7.86 ± 2.99	
Mildly reduced	20	9.90 ± 3.67	
Moderately reduced	24	10.13 ± 3.86	
Severely reduced	13	9.92 ± 3.93[a]	0.030

ANOVA, analysis of variance; RVSWI, right ventricular stroke work index