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Article

Evaluation of Myocardial Work Changes after Lung Resection - The Significance of Surgical Approach: An Echocardiographic Comparison between VATS and Thoracotomy

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Abstract: Considering the controversial benefits of video-assisted thoracoscopic surgery (VATS), we intended to evaluate the impact of surgical approach on cardiac function after lung resection using myocardial work analysis. Echocardiographic data of 45 patients (25 thoracotomy vs. 20 VATS) were retrospectively analyzed. All patients underwent transthoracic echocardiography (TTE) 2 weeks before and after surgery, including two-dimensional speckle tracking and tissue doppler imaging. No notable changes in left ventricular (LV) function, assessed mainly using the LV global longitudinal strain (GLS), global myocardial work index (GMWI), and global work efficiency (GWE), were observed. Right ventricular (RV) TTE values, including tricuspid annular plane systolic excursion (TAPSE), tricuspid annular systolic velocity (TASV), and RV free-wall GLS (RVFWGLS), indicated greater RV function impairment in the thoracotomy group than in the VATS group [TAPSE (mm): 17.90 ± 3.80 vs. 20.60 ± 3.50 , $p < 0.018$; TASV (cm/s): 12.40 ± 2.90 vs. 14.60 ± 2.50 , $p < 0.010$; RVFWGLS (%): -11.50 ± 8.50 vs. -17.50 ± 9.70 , $p < 0.033$], respectively. Unlike RV function, LV function remained preserved after lung resection. The thoracotomy group exhibited greater RV function impairment than did the VATS group. Further studies should evaluate the long-term impact of surgical approach on cardiac function.

Keywords: echocardiography; non-invasive; global myocardial work; thoracotomy; video-assisted thoracoscopic surgery; VATS; lung resection; lung surgery; right ventricle; left ventricle

1. Introduction

Cancer is among the most common causes of death globally, with lung cancer being the foremost cause of cancer-associated death [1,2]. Surgical treatment remains the most common and preferred therapeutic option for early-stage lung cancer [3,4]. Evidence has shown that patients undergoing lung resection through the minimally invasive Video-Assisted Thoracoscopic Surgery (VATS) technique have significantly shorter drainage time and hospital stay and better long-term survival than do patients undergoing conventional lateral thoracotomy [5,6]. There are various levels involved in the complex interdependence between cardiac and respiratory function [7,8]. Lung resections have been associated with long-term cardiorespiratory morbidity, primarily caused by cardiovascular changes resulting from right and LV dysfunction [9,10]. Transthoracic echocardiography (TTE) has

remained the leading method for the routine clinical assessment of cardiac changes after lung resection due to ease of accessibility in standard care hospitals, as well as cost and time effectiveness [11].

The complexity of the right ventricular (RV) anatomy and physiology, especially the marked load dependency of RV function, demands multiparametric echocardiographic assessment, including longitudinal strain, tissue doppler imaging, and myocardial work analysis.[12,13]

The effects of lung resection and surgical approach (i.e., VATS vs. open thoracotomy) on myocardial function have not been adequately studied. Hence, the current study aimed to identify changes in myocardial function after lung resection using myocardial work (MW) analysis, a novel non-invasive method based on echocardiography [14-16].

2. Methods

2.1. Patient collection and study protocol

Our study was conducted at the cardiothoracic surgery department of the university hospital of RWTH Aachen in Germany. Patients who underwent surgery from January 2015 to December 2021 were enrolled (**Figure 1**). We screened 187 patients but only included 45 (25 thoracotomy vs. 20 VATS) owing to exclusion criteria: (1) prior cardiac surgery, (2) prior lung resection, (3) pneumonectomy, (4) severe heart failure, (5) moderate or severe valvular disease, (6) moderate or severe pulmonary hypertension (PAH), (7) inappropriate ultrasound windows, (8) pregnancy, and (9) arrhythmias. Cardio pulmonary bypass and pericardiotomy during cardiac surgery can cause serious changes in right ventricle (RV) and left ventricle (LV) function, consequently we excluded all patients with prior cardiac surgery procedures. [17,18] The patients were then divided into the following two groups: VATS (n = 20) and thoracotomy (n = 25). Our study was based on retrospective patient data collection from our institutional database. Ethical approval was obtained from the Research Ethics Committee of RWTH-University Aachen, Germany (EK 151/09). The ethical board waived the need for informed consent due to the retrospective nature of the study.

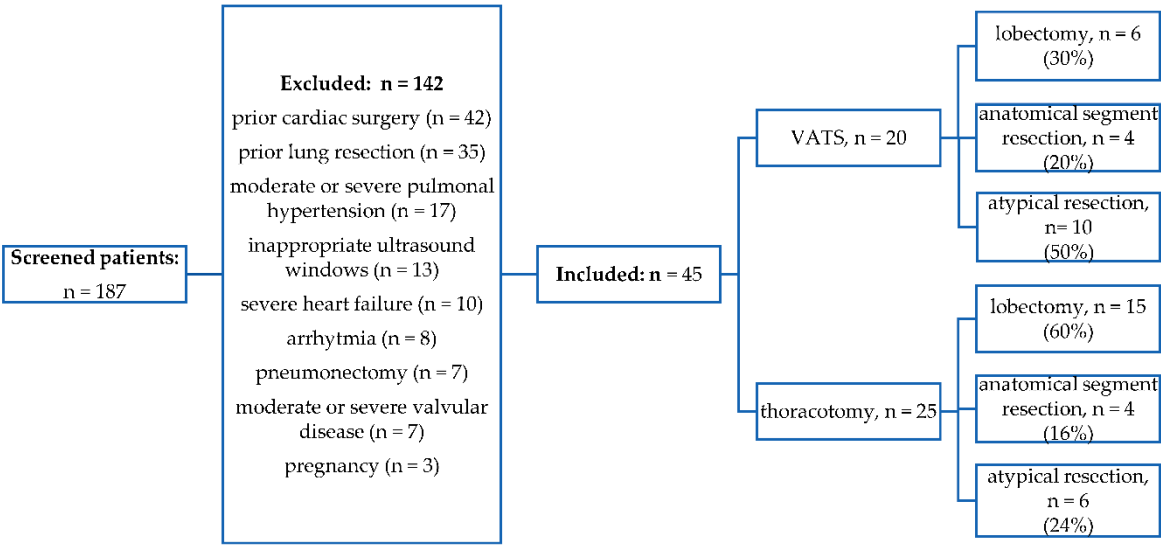


Figure 1. Flow chart of the study: patient collection.

2.2. Echocardiographic measurements

All patients underwent standardized transthoracic echocardiography before and after surgery as part of our routine perioperative clinical care. TTE was performed within 2 weeks before and 2 weeks after the surgery. All echocardiographic measurements were performed in accordance with the European Association of Cardiovascular Imaging and American Society of Echocardiography (ASE) [19-21].

Biplane apical four-chamber (4CH) and two-chamber (2CH) views were used for measuring the left ventricular ejection fraction (LVEF). Peak systolic global longitudinal strain average (GLS avg) from left ventricle (LV) was measured in all three standard apical views, using two-dimensional speckle tracking (2D-STE).

RV diameter and area measurements were made in a modified 4CH view. Right ventricular fractional area change (RVFAC) was calculated using the following formula: (end-diastolic area – end-systolic area) / end-diastolic area × 100%. Additional parameters of the RV like RV free-wall GLS (RVFWGLS) were measured in a modified 4CH view.

Tricuspid annular plane systolic excursion (TAPSE) was measured using M-mode in the apical modified 4CH view from minimum to maximum excursion. Tricuspid annular systolic velocity (TASV) was measured using pulsed-wave tissue doppler imaging of the free tricuspid annulus in the 4CH view.

MW measurements and calculations, including global work index (GWI) and global work efficiency, were performed with a specific commercially available processing software (GE Vivid E90 with the EchoPAC workstation) as described by Russell et al. [16]. Blood pressure was determined using a brachial cuff immediately before each TTE.

All echocardiography studies were performed using Vivid E9 (GE Vingmed Ultrasound AS, Horton, Norway), and measurements were performed using EchoPAC version BT 202 (GE Vingmed Ultrasound AS) (Figure 2).

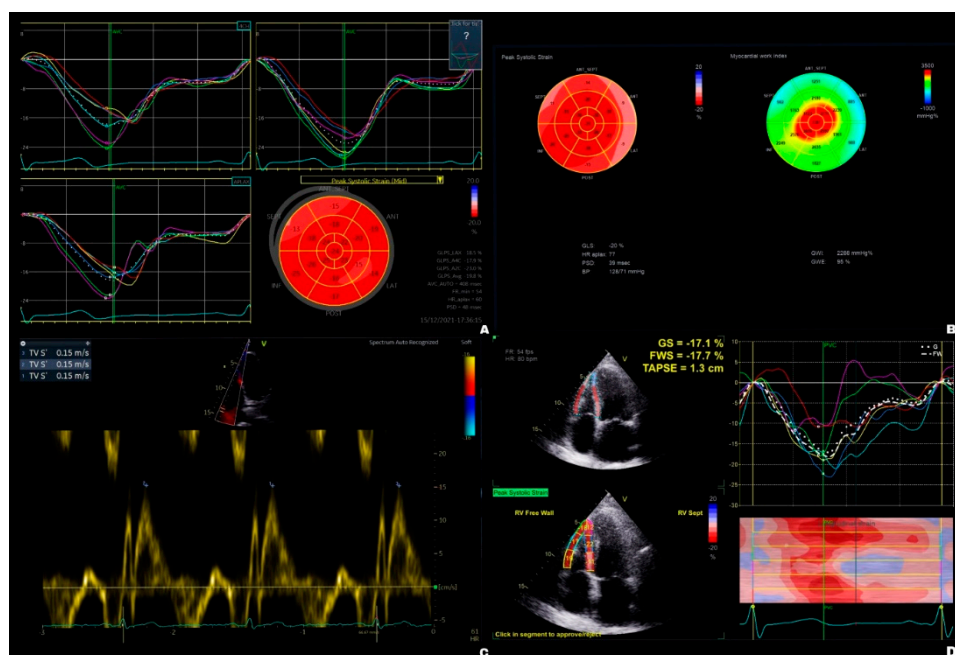


Figure 2. Exemplary demonstration of selected echocardiography studies. (A) GLS of the LV, measured in all three standard apical views; (B) peak systolic strain and GMWI (bull's eye) estimation; (C) TASV estimated from TDI; and (D) GLS of the RV measured in the 4CH view. GLS, global longitudinal strain; LV, left ventricle; GMWI, global myocardial work index; TASV, tricuspid annular systolic velocity; TDI, tissue doppler imaging; RV, right ventricle; 4CH, four-chamber.

2.3. Surgical procedures

In accordance with our hospital's surgical standards, all thoracic procedures were performed under general anesthesia using single-lung ventilation and a double-lumen tube. All patients were placed in right or left lateral decubitus position. For postoperative pain management, epidural or serratus plane nerve catheters were used.

Muscle-sparing antero-lateral thoracotomy in the fifth or sixth intercostal space (ICS) was performed as the conventional surgical approach. In contrast, VATS was performed as the minimally invasive surgical approach, which involved placing one or two 1–2 cm ports at the 7th or

8th ICS following the midaxillary line and a 3rd port at the same ICS level following the posterior axillary line. In cases requiring the use of the uniportal technique, the incision was made at 6th or 7th ICS following the midaxillary line.

2.4. Laboratory tests

Standard perioperative blood tests, including creatinine, hemoglobin, leucocyte, thrombocyte, lactate dehydrogenase (LDH), aspartate transaminase (AST), and alanine transaminase (ALT) levels, were conducted 1 day before and after surgery.

2.5. Statistical analysis

Categorical variables were expressed as absolute numbers and percentages, whereas continuous variables were expressed as means and standard deviations. All analyses were conducted using R statistics inside the R Studio interface version 4.0.3 (RStudio Team, Boston, MA) in conjunction with the jamovi project (2020) (jamovi computer software, version 2.3.9; JAMovi.org), which may be accessed at <https://www.jamovi.org/>. Comparisons between groups were conducted using two-tailed Student's t-tests for normally distributed, non-repeated continuous variables. These tests were run with the assumption that the variables were not identical. Two-way analysis of variance was used to examine continuous repeating variables for intra- and intergroup comparisons. We used either the chi square test or Fisher's exact test, where suitable, for the analysis of categorical variables. Moreover, p values were rounded off to three decimal places or presented as numbers with at least one non-zero digit. A p value of <0.05 indicated statistical significance. A post-hoc power analysis was conducted using G * Power3 and tested the difference between two independent group means using a two-tailed test for the result TASV, a medium effect size ($d = 0.50$) and an alpha of 0.05. [22] The result showed that our study achieves a power of 0.78 with our sample size (thoracotomy $n = 25$ vs. VATS $n = 20$).

3. Results

All procedures were performed uneventfully. No significant adverse events or in-hospital mortality occurred. No vasoactive or inotrope drugs were required postoperatively. After screening a total of 187 patients, 142 were subsequently excluded (Figure 1) The final group consisted of 45 patients, among whom 20 (44.4%) underwent VATS and 25 (55.6%) underwent thoracotomy. Lobectomy, anatomical segment resection, and atypical resection were performed in 6 (30%), 4 (20%), and 10 (50%) patients in the VATS group and 15 (60%), 4 (16%), and 6 (24%) patients in the thoracotomy group, respectively (Figure 1). Table 1 summarize the detailed demographic data for the VATS and thoracotomy groups. The two groups were quite similar in age, body mass index (BMI), sex distribution, preoperative comorbidities, laboratory parameters, and predicted in-hospital death rate (Thoracscore) [23]. The thoracotomy and VATS groups had a mean age of 63 ± 14 and 70 ± 11 years and included 10 and 9 females, respectively.

Table 1. Patient demographics.

	Thoracotomy (n = 25)	VATS (n = 20)	p values
Mean age, years	63 ± 14	70 ± 11	0.10
Female, n (%)	10 (40)	9 (45)	0.74
COPD \geq GOLD III, n (%)	2 (8)	0 (0)	0.06
PAD, n (%)	3 (12)	1 (5)	0.62
Diabetes mellitus, n (%)	3 (12)	6 (20)	0.16
Hypertension, n (%)	17 (68)	15 (75)	0.61
Smoking, n (%)	15 (60)	8 (40)	0.18
KDIGO \geq G3, n (%)	3 (12)	3 (15)	0.37
NYHA \geq III, n (%)	3 (12)	1 (5)	0.59

Non-cardiopulmonary prior thoracic surgery, n (%)	3 (12)	0 (0)	0.49
BMI (kg/m ²)	26.9 ± 4.12	25.2 ± 4.36	0.13
Thoracscore PPD, (%)	2.67 ± 2.75	2.67 ± 2.52	0.87
Creatinine (mg/dL)	0.96 ± 0.41	0.88 ± 0.2	0.85
Hemoglobin (mg/dL)	13.00 ± 1.91	13.70 ± 1.36	0.59
Leukocytes (10 ³ /μL)	7.30 ± 2.82	8.05 ± 3.28	0.27
Thrombocytes (10 ³ /μL)	246 ± 47.2	276 ± 92.1	0.13
LDH (U/L)	198 ± 30.9	210 ± 64.7	0.74
AST (U/L)	23 ± 14.7	24 ± 5.39	0.88
ALT (U/L)	19 ± 16.4	18 ± 7.29	0.57

VATS, video-assisted thoracoscopic surgery; COPD, chronic obstructive pulmonary disease; GOLD, Global Initiative for Chronic Obstructive Lung Disease; PAD, peripheral arterial disease; Diabetes mellitus included patients with oral antidiabetic drugs or insulin therapy; KDIGO, Kidney Disease Improving Global Outcomes; NYHA, New York Heart Association; BMI, body mass index; PPD, Predicted perioperative death rate; LDH, lactate dehydrogenase; AST, aspartate transaminase; ALT, alanine transaminase. Bold entries indicate significance.

3.1. Laboratory blood tests

Hemoglobin levels significantly decreased in both groups after surgery (Thoracotomy: 13.20 ± 1.91 vs. 11.30 ± 1.82 mg/dL; $p < 0.001$; VATS: 13.40 ± 1.36 vs. 12.00 ± 1.74 mg/dL; $p < 0.001$). However, only the thoracotomy group showed a significant increase in leukocytes (8.04 ± 2.82 vs. 9.98 ± 2.74 10³/μL; $p < 0.007$). No discernible difference in any blood test was observed between the groups (Table 2).

Table 2. Comparison of laboratory blood tests between the groups.

	Thoracotomy (n = 25)			VATS (n = 20)			VATS vs. thoracotomy postoperative
	Preoperative	Postoperative	<i>p</i> values	Preoperative	Postoperative	<i>p</i> values	<i>p</i> values
Creatinine (mg/dL)	1.00 ± 0.41	1.02 ± 0.76	0.989	0.92 ± 0.20	0.84 ± 0.23	0.076	0.328
Hemoglobin (mg/dL)	13.20 ± 1.91	11.30 ± 1.82	<0.001	13.40 ± 1.36	12.00 ± 1.74	<0.001	0.631
Leukocytes (10 ³ /μL)	8.04 ± 2.82	9.98 ± 2.74	0.007	8.89 ± 3.28	9.51 ± 4.42	0.766	0.668
Thrombocytes (10 ³ /μL)	249.00 ± 47.20	224.00 ± 82.10	0.236	297.00 ± 92.00	224.00 ± 98.90	0.087	0.175
LDH (U/L)	206.00 ± 30.90	209.00 ± 50.60	0.992	220.00 ± 64.70	210.00 ± 41.80	0.871	0.944
AST (U/L)	26.40 ± 14.70	26.30 ± 13.00	1.000	23.50 ± 5.39	31.90 ± 8.40	0.663	0.576
ALT (U/L)	24.90 ± 16.40	24.30 ± 13.60	0.992	20.10 ± 7.29	19.50 ± 11.20	0.994	0.214

VATS, video-assisted thoracoscopic surgery; LDH, lactate dehydrogenase; AST, aspartate transaminase; ALT, alanine transaminase. **Bold entries indicate significance.**

3.2. Echocardiographic findings within the groups

Table 3 shows the echocardiographic parameters within the groups. No significant changes in all measured echocardiographic parameters, except for TAPSE, were observed within the groups after the surgery. Accordingly, the thoracotomy group (n = 25) showed a significant decrease in TAPSE after the surgery (20.80 ± 3.25 vs. 17.90 ± 3.83 mm; $p < 0.005$; Table 3).

Table 3. Comparison of echocardiographic changes within the groups.

Thoracotomy (n = 25)			VATS (n = 20)		
Preoperative	Postoperative	<i>p</i> values	Preoperative	Postoperative	<i>p</i> values

LVIDd (cm)	4.44 ± 0.55	4.84 ± 0.74	0.101	4.43 ± 0.82	4.53 ± 0.90	0.961
LVIDs (cm)	3.20 ± 0.74	3.50 ± 0.84	0.343	3.18 ± 0.92	3.31 ± 0.93	0.883
LVEDV (mL)	103.00 ± 17.90	106.00 ± 15.20	0.991	101.00 ± 20.30	100.00 ± 22.20	0.992
LVESV (mL)	41.80 ± 11.10	45.20 ± 11.30	0.832	45.00 ± 13.70	45.10 ± 12.90	1.000
LVEF (%)	59.40 ± 6.26	59.2 ± 7.43	0.997	56.3 ± 7.04	56.50 ± 7.70	0.994
CO (L/min)	5.31 ± 1.19	5.66 ± 1.30	0.606	4.90 ± 1.23	4.97 ± 1.09	0.963
GLSLV avg. (%)	-17.40 ± 4.14	-16.7 ± 4.13	0.813	-17.5 ± 2.78	-16.10 ± 4.46	0.532
GWILV	1617 ± 529	1637 ± 462	0.997	1778 ± 580	1505 ± 636	0.074
GWELV (%)	90.90 ± 9.05	90.60 ± 7.56	0.991	93.20 ± 5.72	89.30 ± 10.70	0.068
RVIDd (cm)	3.13 ± 0.63	3.02 ± 0.67	0.674	2.90 ± 0.45	2.81 ± 0.65	0.926
RVIDs (cm)	1.85 ± 0.46	1.90 ± 0.51	0.968	1.64 ± 0.40	1.71 ± 0.41	0.441
RVD1 _{basal} (cm)	3.89 ± 0.79	3.69 ± 0.73	0.714	3.79 ± 0.63	3.81 ± 0.72	0.992
RVD3 _{long} (cm)	6.92 ± 0.79	7.20 ± 0.59	0.356	6.87 ± 0.97	6.81 ± 0.86	0.923
RVFAC (%)	51.2 ± 9.62	51.40 ± 7.55	0.997	47.90 ± 10.60	48.20 ± 7.33	0.994
PASP (mm Hg)	21.00 ± 8.42	21.00 ± 9.01	0.992	16.90 ± 6.59	19.70 ± 9.38	0.482
TAPSE (mm)	20.80 ± 3.25	17.90 ± 3.83	0.005	20.60 ± 3.79	20.60 ± 3.51	1.000
TASV (cm/s)	13.90 ± 3.45	12.40 ± 2.86	0.184	14.10 ± 2.25	14.60 ± 2.54	0.874
RV4CHGLS (%)	-16.80 ± 3.28	-16.00 ± 4.46	0.911	-16.70 ± 6.46	-16.40 ± 4.91	0.992
RVFWGLS (%)	-17.20 ± 9.07	-11.50 ± 8.50	0.133	-18.60 ± 10.30	-17.50 ± 9.71	0.983

VATS, video-assisted thoracoscopic surgery; LVIDd, Left ventricular internal diameter end diastole; LVIDs, Left ventricular internal diameter end systole; LVEDV, Left ventricular end-diastolic volume; LVESV, Left ventricular end-systolic volume; LVEF, left ventricular ejection fraction; CO, cardiac output; GLSLVavg., global longitudinal strain left ventricular average; GWI LV, global work index left ventricle; GWELV, global work efficiency left ventricle; RVIDd, right ventricular internal diameter at end diastole; RVIDs, right ventricular internal diameter at end systole; RVD1 basal, right ventricular basal diameter at end-diastole; RVD3 long, right ventricular longitudinal diameter at end-diastole; RVFAC, right ventricular fractional area change; PASP, pulmonary arterial systolic pressure; TAPSE, tricuspid annular plane systolic excursion; TASV, tricuspid annular systolic velocity; RV4CHGLS, right ventricular four-chamber global longitudinal strain; RVFWGLS, right ventricular free-wall global longitudinal strain. **Bold entries indicate significance.**

3.3. Echocardiographic findings between groups

On the other hand, significant changes in postoperative RV echocardiography parameters indicating impairment in RV function were found between thoracotomy and VATS groups (TAPSE 17.90 ± 3.80 vs. 20.60 ± 3.50 mm: p < 0.018; TASV: 12.40 ± 2.90 vs. 14.60 ± 2.50 cm/s: p < 0.010; RVFWGLS: -11.50% ± 8.50% vs. -17.50% ± 9.70%; p < 0.033; **Table 4**).

Table 4. Comparison of echocardiographic changes between the groups.

	Thoracotomy	VATS	p values
preoperative			
LVIDd (cm)	4.40 ± 0.60	4.40 ± 0.80	0.975
LVIDs (cm)	3.20 ± 0.70	3.20 ± 0.90	0.921
LVEDV (mL)	102.70 ± 17.90	100.60 ± 20.30	0.712
LVESV (mL)	41.80 ± 11.10	45.00 ± 13.70	0.391
LVEF (%)	59.40 ± 6.30	56.30 ± 7.00	0.130
CO (L/min)	5.30 ± 1.20	4.90 ± 1.20	0.264
LVGLS (%)	-17.40 ± 4.10	-17.50 ± 2.80	0.922
LVGWI	1616.80 ± 529.50	1778.00 ± 580.00	0.347
LVGWE (%)	90.90 ± 9.10	93.20 ± 5.70	0.344
RVIDd (cm)	3.10 ± 0.60	2.90 ± 0.40	0.178
RVIDs (cm)	1.80 ± 0.50	1.60 ± 0.40	0.135

RVD1 _{basal} (cm)	3.90 ± 0.80	3.80 ± 0.60	0.625
RVD3 _{basal} (cm)	6.90 ± 0.80	6.90 ± 1.00	0.823
RVFAC (%)	51.20 ± 9.60	47.90 ± 10.60	0.283
PASP (mm Hg)	21.00 ± 8.40	16.90 ± 6.60	0.095
TAPSE (mm)	20.80 ± 3.20	20.60 ± 3.80	0.857
TASV (cm/s)	13.90 ± 3.50	14.10 ± 2.20	0.835
RV4CHGLS (%)	-16.80 ± 3.30	-16.70 ± 6.50	0.957
RVFWGLS (%)	-17.20 ± 9.10	-18.60 ± 10.30	0.647
postoperative			
LVIDd (cm)	4.80 ± 0.70	4.50 ± 0.90	0.228
LVIDs (cm)	3.50 ± 0.80	3.30 ± 0.90	0.481
LVEDV (mL)	105.90 ± 15.20	100.00 ± 22.20	0.313
LVESV (mL)	45.20 ± 11.30	45.10 ± 12.90	0.993
LVEF (%)	59.20 ± 7.40	56.50 ± 7.70	0.249
CO (L/min)	5.70 ± 1.30	5.00± 1.10	0.072
LVGLS (%)	-16.70 ± 4.10	-16.10 ± 4.50	0.670
LVGWI	1636.80 ± 462.00	1504.90 ± 636.40	0.448
LVGWE (%)	90.60 ± 7.60	86.30 ± 10.60	0.141
RVIDd (cm)	3.00 ± 0.70	2.80 ± 0.60	0.329
RVIDs (cm)	1.90 ± 0.50	1.70 ± 0.40	0.212
RVD1 _{basal} (cm)	3.70 ± 0.70	3.80 ± 0.70	0.597
RVD3 _{basal} (cm)	7.20 ± 0.60	6.80 ± 0.90	0.093
RVFAC (%)	51.40 ± 7.50	48.20 ± 7.30	0.174
PASP (mm Hg)	21.00 ± 9.00	19.70 ± 9.40	0.664
TAPSE (mm)	17.90 ± 3.80	20.60 ± 3.50	0.018
TASV (cm/s)	12.40 ± 2.90	14.60 ± 2.50	0.010
RV4CHGLS (%)	-16.00 ± 4.50	-16.40 ± 4.90	0.787
RVFWGLS (%)	-11.50 ± 8.50	-17.50 ± 9.70	0.033

VATS, video-assisted thoracoscopic surgery; LVIDd, Left ventricular internal diameter end diastole; LVIDs, Left ventricular internal diameter end systole; LVEDV, Left ventricular end-diastolic volume; LVESV, Left ventricular end-systolic volume; LVEF, left ventricular ejection fraction; CO, cardiac output; GLSLVavg., global longitudinal strain left ventricular average; GWILV, global work index left ventricle; GWELV, global work efficiency left ventricle; RVIDd, right ventricular internal diameter at end diastole; RVIDs, right ventricular internal diameter at end systole; RVD1 basal, right ventricular basal diameter at end-diastole; RVD3 long, right ventricular longitudinal diameter at end-diastole; RVFAC, right ventricular fractional area change; PASP, pulmonary arterial systolic pressure; TAPSE, tricuspid annular plane systolic excursion; TASV, tricuspid annular systolic velocity; RV4CHGLS, right ventricular four-chamber global longitudinal strain; RVFWGLS, right ventricular free-wall global longitudinal strain. **Bold entries indicate significance.**

4. Discussion

The main findings of the present study suggest that RV function deteriorated more significantly after thoracotomy than after VATS. This is shown by the significant decrease in TAPSE, TASV, and RVFWGLS in the thoracotomy group (**Table 4**). Modifications in RV function were noted despite preservation of LV function, suggesting that the RV/pulmonary vascular unit was the primary target of modifications after lung resection. We showed that pulmonary resection during thoracic surgery (either open or minimally invasive surgery) had no major effect on RV or LV diastolic or systolic function. However, VATS promoted better preservation of RV function than did thoracotomy. In

both thoracotomy and VATS groups, no major RV dysfunction following pulmonary resection was observed. Conversely, Reed et al. [24], who did observe major RV dysfunction, concluded that this dysfunction was multifactorial and may solely be attributed to a contractile impairment of the RV. The discrepancy between our results and those shown by Reed et al. [24] may be explained by the fact that we analyzed the postoperative echocardiogram data 2 weeks after the surgery, whereas Reed et al. [24] analyzed the data during the very early postoperative period (postoperative days 1 and 2). A lung resection can cause a reduction in the pulmonary vascular system [25]. This, in turn, can lead to a reduction in the pulmonary vascular cross-sections, which consequently results in pulmonary arterial hypertension and then right heart failure. During thoracotomy and anesthesia, alterations occur in the pulmonary vascular system. Additionally, evidence has shown a rise in intrathoracic pressures [26], which is detrimental to the filling pressure of the RV [25,27]. On the other hand, neither the systolic RV parameters nor RV inflow showed any signs of an identifiable alteration. We hypothesize that the RV afterload would greatly increase in the acute phase after lung resection and that the RV function would deteriorate in the very early postoperative period as the heart struggles to adjust to the increasing afterload. However, after comparing the postoperative RV echocardiographic parameters between the VATS and thoracotomy groups, we found that the VATS group had better RV function preservation than did the thoracotomy group. One possible explanation for this finding could be the sudden establishment of a negative intrathoracic pressure, which has the potential to severely compromise ventricular mechanics [28]. This phenomenon occurs more frequently during abnormal breathing in patients undergoing thoracotomy than in those undergoing VATS due to more severe postoperative pain [29,30].

Our study has been the first to use the novel, non-invasive MW assessment of the LV function after lung resection. MW is a new method for measuring LV performance that has lesser reliance on load than does GLS [31,32]. An increase in preload or afterload may decrease strain while maintaining MW under various physiologic and pathologic situations. MW enables the measurement of global function after adjusting for systolic blood pressure. Despite the independency of the MW from pre- to afterload, we detected no significant changes in the LVMWI or MWE after lung resection in either the thoracotomy or VATS group. However, non-invasive measurements of the RVMW have still not been available, and we were not able to perform RVMW analysis. Examination of RV pressure-volume loops before and after surgery following lobar pulmonary artery clamping convincingly showed an impairment in patients after lung resection under experimental settings [25,33]. Despite this drastic redistribution of pulmonary blood flow during lung resection, one study showed that systemic arterial blood pressure and cardiac output were maintained in the great majority of patients undergoing thoracic surgery even with lobectomy [27]. In a few of individuals, however, pulmonary vascular or RV comorbidity may hinder their ability to adjust appropriately [25,27].

5. Conclusions

Our results showed that LV function was preserved after lung surgery. In contrast, thoracotomy promoted a significantly greater deterioration in RV function than did VATS. Pain associated with abnormal breathing mechanics may have a negative impact on ventricular filling and contractility. This should be investigated prospectively in larger cohorts with longer follow-up durations.

6. Limitations

This study suffers from the typical shortcomings associated with a single-center study. Our study included a small number of patients, which, along with the retrospective analysis, limits the interpretation of our results. Given that none of the TTE measurements were particularly taken for the intended study but rather for clinical examinations as part of regular perioperative care protocols, the echocardiography examination time frames were not similar. Given the retrospective nature of our study, patient selection and data collection were susceptible to potential bias. The limited number of patients in our study did not allow us to conduct a sub group analysis to explore the impact of the resected lung volume on the RV and LV function changes.

The complexity of the RV anatomy and physiology, particularly the marked load dependence of RV function, also contributes to measurement variation. However, we excluded individuals with any cardiac structural or valvular disease given that inaccurate LV pressure calculation based on brachial-cuff blood pressure measurements may arise owing to individual anatomical or pathological variations in the artery system.

Given that our study had a maximum follow-up duration of 2 weeks, more research with larger cohorts and longer-term follow-up would be required to make any long-term conclusions. However, due to the fact that all of our patients are oncology patients, and the majority of them required chemotherapy and / or radiation after surgery, and due to the cardiotoxicity of chemotherapy, we were unable to conduct long-term echocardiography studies because it will be impossible to distinguish the cause of the further changes in LV and RV (cardiotoxicity or surgery) in long-term follow-up.

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Data Availability Statement: The data presented in this study are available on request from the corresponding author.

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