Is Systolic Right Ventricular Function Reduced after Thoracic Non-Cardiac Surgery? A Propensity Matched Echocardiographic Analysis

Henning Johann Steffen, MD,† Sebastian Kalverkamp, MD,† Rashad Zayat, MD,† Rüdiger Autschbach, MD, PhD, Jan-Wilhelm Spillner, MD, PhD, Andreas Hagendorff, MD, PhD, and Nima Hatam, MD

Purpose: To assess whether thoracic non-cardiac surgery has an influence on right ventricular function (RVF) compared to known impaired postoperative RV function after cardiac surgery.

Methods: In all, 50 patients (mean age: 61 years), who underwent thoracic non-cardiac surgery were included and matched using propensity score to 50 patients, receiving coronary artery bypass graft surgery (CABG) (CABGmatched). All patients had transthoracic echocardiography (TTE) including two-dimensional speckle tracking (2D-STE) and tissue Doppler imaging (TDI) pre- and 1 week postoperatively.

Results: No significant changes in RV measurements including tricuspid annular plane systolic excursion (TAPSE), tricuspid annular systolic velocity (TASV), RV fractional area change (RV-FAC), and 2D-STE of the RV and RV freewall within the thoracic non-cardiac surgery patients comparing pre- and postoperative values. Comparing RV TTE values between CABGmatched patients and thoracic surgery patients, only TAPSE differed between groups preoperatively (p < 0.0001), where postoperatively, all RV measurements differed significantly between the two groups: TAPSE (p < 0.0001), TASV (p < 0.001), RVFAC (p = 0.005), and RV 2D-STE (p < 0.0001) indicating impairment of RV function post-CABG surgery compared to thoracic non-cardiac surgery.

Conclusion: Thoracic non-cardiac surgery including an opening of the pleural cavity did not influence RV function early postoperative, whereas CABG surgery with pericardiotomy led to an impaired global RV function.

Keywords: right ventricular function, thoracic non-cardiac surgery, coronary artery bypass grafting, echocardiography, strain analyses

Introduction

The non-invasive assessment of systolic right ventricular function (RVF) has gained increasing evidence in clinical practice and plays a central role in various clinical scenarios including ischemic heart disease, cardiomyopathies, and especially in the management of pulmonary artery hypertension (PAH). Aside of these diseases, RVF is an important aspect while examining and treating patients after cardiac surgery, in particular after coronary artery bypass graft surgery (CABG). Previous studies
have shown that due to cardiac surgery, RVF is a crucial determinant of the postoperative outcome.\textsuperscript{6-10} Cardiac magnetic resonance (cMR) imaging remains the gold standard in the assessment of RV morphology (e.g., volumes and diameters) and function, particularly via ejection fraction (EF). In spite of its diagnostic value, several technical and infrastructural limitations still prevail. To name a few, a maximum frame rate of 35 images per second, immense costs in operations and maintenance, staff resource-intensity, and/or missing availability in non-maximum-care hospitals.\textsuperscript{1,3,11} Two-dimensional (2D) transthoracic echocardiography (TTE) has evolved technically and costily over the past decade and is now almost ubiquitously available in daily clinical routine.\textsuperscript{3,12-15} Regarding quantification of systolic RVF, standard TTE is not able to deliver the same values to the intricacy of RV geometry compared to the left ventricle (LV). Three-dimensional echocardiography somewhat appears to be the technical solution for quantifying RVF by EF, but the downside being dependent on excellent image quality, which is hardly achieved in clinical practice, especially after cardiac surgery. Surrogate parameters like tricuspid annular plane systolic excursion (TAPSE) or RV fractional area change (RV-FAC) fairly approximate RVF compared to cMR.\textsuperscript{1,3} In addition, recent parameters derived by tissue Doppler imaging (TDI) via tricuspid annular systolic velocity (TASV) or deformation imaging using two-dimensional speckle tracking (2D-STE) to quantify global longitudinal strain (GLS), and strain rate (SR).\textsuperscript{16,17} The deterioration of TASPE and TASV after cardiac surgery, especially after opening the pericardial cavity, is a familiar acute and long-term circumstance and is significant.\textsuperscript{5,6,8-10} Only two earlier studies showed the effects of thoracic surgery on systolic right heart function.\textsuperscript{4,18} Furthermore, the knowledge about changes in RVF after cardiac and thoracic surgery is still somewhat incomplete.

The aim of our study was to evaluate whether the opening of the chest in thoracic surgery procedures, either as an open surgery via lateral thoracotomy or minimal-invasive via video-assisted thoracoscopic surgery (VATS), affects RV- and LV-function postoperatively, in comparison to the RVF in patients undergoing coronary artery bypass surgery (CABG) using 2D-TTE.

**Methods**

**Patient collective and study protocol**

After approval from our local ethic committee (Ethik-Kommission an der medizinischen Fakultät der RWTH Aachen, EK: 151/09), all patients who underwent thoracic, non-cardiac, surgery (TS) at our institute were screened from January 2016 till December 2016. Exclusion criteria for the TS group were as follows: 1) prior cardiac surgery, 2) atrial fibrillation, 3) coronary artery disease, 4) heart failure in medical history, 5) valvular heart disease, 6) inappropriate ultrasound windows, 7) moderate or severe tricuspid insufficiency, and 8) patient participated in other study. In all, 50 patients were included, and informed consent was obtained from each enrolled patient. As control group, we matched all patients in the TS group to CABG patients from our historical control-cohort (120 patients).

**Echocardiographic measurements**

All patients underwent preoperative and postoperative standardized TTE. TTE was performed the day before operation and on the 7th postoperative day (POD). A complete standard M-mode and 2D echocardiographic exam including tissue-Doppler (TDI) and 2D-STE was performed according to the guidelines of the European Association of Cardiovascular Imaging (EACVI) and American Society of Echocardiography (ASE).\textsuperscript{19,20} Biplane EF of the LV was measured using the Simpsons method from apical four (4CH)- and two-chamber (2CH) views. Peak systolic global longitudinal strain average (GLS avg) from LV was measured from all three apical views. Pulsed-wave-Doppler (PWD) of the mitral-valve inflow was recorded quantifying E- and A-wave velocity. É-wave velocity was measured using PWD-TDI of the septal and lateral mitral-valve annulus. RV diameter and area were measured in the modified 4CH. RV-FAC was calculated as (RV end-diastolic area (RVEDA) − RV end-systolic area ((RVESA)/RVEDA × 100). PWD measurement of the tricuspid inflow included E- and A-wave velocity. PWD-TDI of the free tricuspid annulus in the 4CH view was used to record É-wave velocity and TASV in cm/s. To evaluate TAPSE, the M-mode-slope of the tricuspid lateral annulus measured from the onset of QRS complex to its maximum excursion at end systole. GLS and SR from the RV were measured in the modified 4CH (Fig. 1). In addition, the GLS and SR of the RV freewall were separately measured (Fig. 1). All echocardiography studies were performed using the Vivid E9 (GE Vingmed Ultrasound AS, Horten, Norway) and the measurements were done with EchoPAC version BT 113 (GE Vingmed Ultrasound AS, Horten, Norway).
Surgical procedures

**CABG surgery**

All patients underwent cardiac surgery through full median sternotomy. Procedures were performed on-pump using cardio pulmonary bypass. Myocardial protection was ensured through antegrade crystalloid cardioplegia with mild hypothermia (32–34°C). As our institutional standard, pericard was left open after CABG surgery.

**Thoracic non-cardiac surgery**

All thoracic procedures were performed under general anesthesia, and patients were intubated with a double-lumen tube for one-lung ventilation and positioned in a lateral decubitus position. An epidural catheter was positioned for postoperative analgesia. Procedures performed were as follows: atypical (non-anatomical) lung resections (n = 14, 28%), anatomical lobectomy (n = 21, 42%) or bi-lobectomies (n = 11, 22%), pleurolysis, pleurectomy, or tissue sampling (n = 4, 8%). Wounds were closed with absorbable and non-absorbable sutures.

**Open thoracotomy**

All operations were performed in the same manner. Using standard protocols, muscle-sparing antero- or posterolateral thoracotomy were performed abreast the fifth intercostal space.

**Video-assisted thoracoscopic surgery**

VATS were performed using a uni- (n = 5, 29%) or multiportal (n = 12, 71%) technique. In uniportal VATS, the sixth intercostal space of the anterior axillary line was incised. For a multiportal VATS, two 1- to 2-cm ports were brought in at the level of the seventh intercostal space of the mid-axillary line and the lateral scapular line. A minithoracotomy of 2–3 cm was performed between the fourth or fifth intercostal space of the anterior axillary line. Standard thoracoscopic instruments were used and the whole situs visualized during operation procedure.

**Statistical analysis**

Continuous variables are expressed as mean ± standard deviation (SD) and categorical variables as absolute
numbers and percentages. Data analysis was performed with SPSS 23 (IBM, Chicago, IL, USA). Propensity scores were calculated for each patient in the thoracic surgery group (n = 50) and patients in our historical control cohort with CABG patients (n = 120) using multivariate logistic regression based on the following preoperative covariates: Age, gender, EF, and body mass index (BMI). TS patients were matched to the CABG patients with the closest propensity score with the nearest-neighbor algorithm without replacement and with a 0.2 matching tolerance.

Continuous repeated variables were analyzed with two-way ANOVA for the comparison between and within groups. Categorical variables were analyzed with a chi-square test or, if appropriate, Fisher’s exact test, for the comparison between non-matched groups. McNemar test was used to compare categorical variables between TS patients and the matched CABG patients (CABGmatched). p values were reported as three digit numbers or with at least one non-zero digit. A p value < 0.05 was considered statistically significant.

**Results**

All patients in the TS and CABGmatched groups had uneventful surgery. There were no serious adverse events and no in-hospital deaths. No relevant tricuspid regurgitation occurred postoperatively. In all, 18 patients in CABGmatched group had revascularization of their right coronary. In the TS group surgery, 33 patients had a lateral thoracotomy (Thoracotomy group) and 27 patients had minimal invasive surgery (VATS). In the TS group, 11 patients had bi-lobectomy surgery, 21 patients underwent uni-lobectomy procedures, and 11 patients had atypical lung resection (Table 1).

Detailed demographic data of all TS and CABGmatched groups are presented in Table 1. Mean age of the TS patients was 61.2 ± 12.4 years including 21 females. Mean patients age in the CABGmatched group was 64.5 ± 8.5 years including 19 females. The matched group did differ in many known risk factors such as peripheral artery disease (PAD), nicotine abuse, and insulin-dependent diabetes mellitus (IDDM) as presented in Table 1.

**Echocardiographic findings within the groups**

**Thoracic surgery patients**

Within the thoracic surgery group (n = 50), no significant changes could be detected in any measured RV and LV parameters postoperatively compared to preoperatively (Table 2).

**Subgroup VATS**

Also in patients, who underwent minimal-invasive thoracic surgery via VATS, RV, and LV echo parameters

| Table 1 Patient demographics |
|-----------------------------|
|                           | Thoracic (n = 50) | CABG matched (n = 50) | p values | VATS (n = 17) | Thoracotomy (n = 33) | p values |
| Mean age (y)               | 61.2 ± 12.4       | 64.5 ± 8.5           | 0.103    | 59.69 ± 16.0 | 61.78 ± 10.5        | 0.617   |
| Female, n (%)              | 21 (42.0)         | 19 (38)              | 0.193    | 8 (47.1)     | 13 (39.4)           | 0.763   |
| BMI (kg/m²)                | 25.8 ± 4.4        | 25.2 ± 6.8           | 0.618    | 26.6 ± 4.3   | 25.0 ± 4.5          | 0.292   |
| COPD, n (%)                | 10 (20.0)         | 6 (12)               | 0.001    | 1 (5.9)      | 9 (27.3)            | 0.133   |
| CAD, n (%)                 | 8 (16.0)          | 50 (100.0)           | 0.008    | 3 (17.7)     | 5 (15.2)            | 0.999   |
| Creatinine (mg/dl)         | 1.1 ± 0.3         | 1.0 ± 0.3            | 0.098    | 1.0 ± 0.2    | 1.0 ± 0.3           | 0.627   |
| Hypertension n (%)         | 22 (44.0)         | 26 (52)              | 0.254    | 7 (41.2)     | 15 (45.5)           | 0.999   |
| IDDM, n (%)                | 6 (12.0)          | 8 (16.0)             | 0.011    | 2 (11.8)     | 4 (12.1)            | 0.999   |
| Nicotine, n (%)            | 18 (36.0)         | 14 (28.0)            | 0.011    | 5 (29.4)     | 13 (39.4)           | 0.548   |
| Pre-op dialysis n (%)      | 1 (2.0)           | 0 (0.0)              | 0.087    | 0 (0.0)      | 1 (3.1)             | 0.999   |
| PAD, n (%)                 | 3 (6.0)           | 4 (8.0)              | 0.022    | 1 (5.9)      | 2 (6.1)             | 0.999   |
| Atypical lung resection, n (%) | 14 (28)         | -                    | 6 (35.2) | 8 (24.2)     | 0.510                  |
| Lobectomy, n (%)           | 21 (42)           | -                    | 12 (70.6) | 9 (27.2)     | 0.005                  |
| Bi-lobectomy, n (%)        | 11 (22)           | -                    | 1 (5.8)  | 10 (30.3)   | 0.072                  |
| Pleurolyse, pleurodese and PE, n (%) | 4 (8) | - | 4 (23.5) | 0 | 0.010                  |
| 1 CAD, n (%)               | -                 | 3 (6)                | -        | -           | -                     |
| 2 CAD, n (%)               | -                 | 12 (24)              | -        | -           | -                     |
| 3 CAD, n (%)               | -                 | 35 (70)              | -        | -           | -                     |

BMI: body mass index; COPD: chronic obstructive pulmonary disease; CAD: coronary artery disease; IDDM: insulin-dependent diabetes mellitus; PAD: peripheral artery disease, CABG: coronary artery bypass grafting; PE: probe excision; Bold writing indicates significance.
did not differ significantly comparing pre- and postoperative values (Table 3).

**Subgroup thoracotomy**

Even the patients, who received open thoracic surgery with lateral thoracotomy, did not present significant changes in RV and LV TTE parameters postoperatively (Table 3).

**Subgroup CABGmatched**

On the other hand, almost all RV and LV measured parameters changed significantly indicating an impaired RV and LV function postoperatively compared to preoperatively. Only tricuspid valve inflow Doppler (E/A) and the mitral valve (E/ε) did not change in the CABGmatched patients.

**Echocardiographic findings between groups**

**Thoracic surgery patients vs. CABGmatched**

Regarding the preoperative RV echocardiography parameters, only TAPSE and SR of the RV freewall showed significant changes between thoracic surgery patients and the CABGmatched group (TAPSE: 23.7 ± 4.6 vs. 19.3 ± 2.5 mm, p < 0.0001 and RV SR freewall: −1.6 ± 0.6 vs. −1.3 vs. 0.3, p = 0.027), respectively (Table 4). In terms of preoperative functional LV echo parameter, EF did not differ between thoracic surgery patients and CABGmatched group, whereas global peak longitudinal strain average of the LV (LV GLS avg.) was significantly lower in the CABGmatched group compared to the thoracic surgery group (−13.5 ± 4.0 vs. −19.3 ± 4.0, p < 0.0001, respectively). The mitral valve Doppler inflow ratio (MV E/A) was also significantly lower in the CABGmatched group compared to the thoracic surgery group, preoperatively (Table 4).

Postoperatively all RV echo parameters, including 2D-STE and TDI, were significantly inferior in the CABGmatched group compared to the thoracic surgery group, indicating a postoperative impairment of the RV systolic function in the CABGmatched group compared to preserved RV function in thoracic surgery patients (Table 4). Regarding the postoperative LV-EF and LV GLS avg., both parameters were significantly lower in the CABGmatched group compared to the thoracic surgery patients (Table 4). The tricuspid valve Doppler-inflow parameter (TV E/A) and TDI (TV E/ε) were in the normal range in both groups preoperatively, but postoperatively in the CABGmatched group TV E/A decreased and simultaneously TV E/ε increased significantly compared to the thoracic surgery patients (Table 4), which suggests a pseudonormal diastolic filling pattern of the RV in the CABGmatched group equating to second-degree
diastolic dysfunction. The MV inflow parameter did not differ significantly between groups postoperatively.

**Comparison within the thoracic surgery patients comparing wedge resection vs. lobectomy**

To compare RV function after major and minor pulmonary resection, we divided the thoracic patients into two groups regarding the surgery method (lobectomy group (n = 32) vs. wedge resection (n = 16) (atypical lung resection). In the wedge resection group (n = 14), TAPSE was 23.9 ± 3.2 mm and GLS RV 4CH was −20.1 ± 5.7% preoperatively and 23.9 ± 1.5 mm and 19.5 ± 4.4 postoperatively (p = 1.000 and p = 0.742, respectively). In the lobectomy group, TAPSE was 25.8 ± 5.2 mm and GLS RV 4 CH was −21.5 ± 5.8% preoperatively and 24.9 ± 5.1 and −21.4 ± 7.5% postoperatively (p = 0.900 and p = 0.952). Comparing TAPSE and GLS RV 4 CH postoperatively between wedge resection and lobectomy group, no significant differences could be detected (p = 0.803 and p = 0.987, respectively). We could also not detect any significant difference in further RV parameters between the two groups.

**Discussion**

In this study, we found that after thoracic surgery either as open or minimal invasive surgery, there were no changes in diastolic or systolic RV and LV function, whereas a striking reduction in overall RV function after cardic surgery with pericardiectomy was detected. The reduction in many RV parameters, such as TAPSE, after cardiac surgery has been widely recognized, but the definite underlying mechanisms are still to be fully understood. Some studies suggested that the impairment of RV function during cardiac surgery is due to poor myocardial protection, while on cardio-pulmonary bypass. Changes in RV function after thoracic surgery are still unclear. We demonstrated that thoracic surgery, open or minimal invasive surgery, with major pulmonary resection (bi-lobectomy), did not have any influence on diastolic and systolic function of the RV or LV. In contrast to our findings, Reed et al. detected RV dysfunction after pulmonary resection, and concluded that this dysfunction is multifactorial and might not only be due to a contractile impairment of the RV. A possible explanation for this discordance between our findings and the findings demonstrated by Reed et al. is the different selected time point in performing the evaluation of the RV function, while Reed et al. performed the evaluation after the early postoperative period (PODs 1 and 2) and we performed the postoperative echocardiography analysis 1 week postoperatively. We believe that during the operation and the ICU stay many factors will
Table 4  Preoperative and postoperative echocardiographic findings in comparison between groups

|                  | Thoracic (n = 50) | CABG matched (n = 50) | p values | VATS (n = 17) | Thoracotomy (n = 33) | p values |
|------------------|-------------------|-----------------------|----------|--------------|----------------------|----------|
| TAPSE (mm)       | 23.7 ± 4.6        | 19.3 ± 2.5            | <0.0001  | 23.6 ± 5.3   | 24.1 ± 4.6           | 0.937    |
| TASV (cm/s)      | 12.4 ± 3.3        | 12.8 ± 2.9            | 0.748    | 12.2 ± 3.4   | 12.8 ± 4.3           | 0.892    |
| TV E/A           | 1.4 ± 0.5         | 1.2 ± 0.2             | 0.069    | 1.6 ± 0.71   | 1.3 ± 0.4            | 0.279    |
| TV E/é           | 5.6 ± 2.8         | 5.6 ± 1.2             | 0.999    | 5.8 ± 2.4    | 5.6 ± 3.2            | 0.975    |
| RV FAC (%)       | 52.5 ± 13.6       | 54.0 ± 5.9            | 0.818    | 51.3 ± 7.3   | 53.3 ± 12.0          | 0.812    |
| GLS RV 4CH (%)   | −19.9 ± 4.8       | −18.5 ± 4.7           | 0.243    | −19.0 ± 5.5  | −20.5 ± 5.6          | 0.681    |
| GLS RV freewall (%) | −25.1 ± 5.8   | −23.1 ± 5.4           | 0.431    | −23.8 ± 7.0  | −25.4 ± 5.4          | 0.741    |
| RV SR 4CH (1/s)  | −1.1 ± 0.3        | −1.0 ± 0.2            | 0.104    | −1.1 ± 0.3   | −1.2 ± 0.3           | 0.548    |
| RV SR freewall (1/s) | −1.6 ± 0.6     | −1.3 ± 0.3            | 0.027    | −1.7 ± 0.8   | −1.6 ± 0.4           | 0.593    |
| Peak disp. basal (%) |            |                       |          |              |                      |          |
| LVEF (%)         | 59.3 ± 8.2        | 58.9 ± 4.6            | 0.973    | 58.2 ± 8.7   | 59.9 ± 8.0           | 0.785    |
| GLS LV avg. (%)  | −19.3 ± 4.0       | −13.5 ± 4.0           | <0.0001  | −17.8 ± 2.6  | −20.2 ± 4.5          | 0.158    |
| MV E/A           | 1.2 ± 0.6         | 0.8 ± 0.3             | 0.009    | 1.3 ± 0.6    | 1.1 ± 0.5            | 0.262    |
| MV E/é           | 8.5 ± 3.1         | 9.1 ± 3.8             | 0.759    | 8.8 ± 3.8    | 8.3 ± 2.8            | 0.875    |
| Post-op          |                   |                       |          |              |                      |          |
| TAPSE (mm)       | 23.1 ± 4.9        | 10.6 ± 2.4            | <0.0001  | 26.4 ± 6.3   | 23.2 ± 3.8           | 0.121    |
| TASV (cm/s)      | 12.1 ± 3.3        | 9.5 ± 2.1             | <0.001   | 12.5 ± 3.6   | 13.3 ± 3.1           | 0.825    |
| TV E/A           | 1.3 ± 0.4         | 1.1 ± 0.3             | 0.007    | 1.5 ± 0.5    | 1.3 ± 0.4            | 0.445    |
| TV E/é           | 6.2 ± 2.7         | 8.5 ± 3.8             | 0.005    | 6.6 ± 2.4    | 6.2 ± 3.3            | 0.943    |
| RV FAC (%)       | 52.0 ± 8.9        | 44.0 ± 6.4            | 0.005    | 52.7 ± 7.9   | 51.6 ± 9.6           | 0.929    |
| GLS RV 4CH (%)   | −20.3 ± 5.6       | −11.3 ± 2.8           | <0.0001  | −22.0 ± 5.9  | −19.2 ± 5.3          | 0.292    |
| GLS RV freewall (%) | −24.4 ± 7.6     | −14.6 ± 5.2           | <0.0001  | −25.1 ± 8.0  | −23.9 ± 7.3          | 0.851    |
| RV SR 4CH (1/s)  | −1.2 ± 0.3        | −0.7 ± 0.4            | <0.0001  | −1.2 ± 0.3   | −1.2 ± 0.3           | 0.998    |
| RV SR freewall (1/s) | −1.6 ± 0.5     | −1.2 ± 0.3            | 0.007    | −1.6 ± 0.7   | −1.5 ± 0.4           | 0.925    |
| Peak disp. basal (%) |            |                       |          |              |                      |          |
| LVEF (%)         | 60.3 ± 6.8        | 54.6 ± 8.2            | 0.0071   | 62.2 ± 5.8   | 59.2 ± 7.2           | 0.448    |
| GLS LV avg. (%)  | −19.9 ± 4.0       | −10.6 ± 3.1           | <0.0001  | −20.4 ± 3.3  | −19.6 ± 4.3          | 0.816    |
| MV E/A           | 1.1 ± 0.5         | 1.1 ± 0.4             | >0.999   | 1.2 ± 0.5    | 1.0 ± 0.6            | 0.552    |
| MV E/é           | 9.0 ± 3.3         | 9.9 ± 2.9             | 0.541    | 9.3 ± 3.7    | 8.7 ± 3.1            | 0.863    |

VATS: video-assisted thoracoscopic surgery; TAPSE: tricuspid annular plane systolic excursion; TASV: tricuspid annular systolic velocity; TV E/A: tricuspid valve E/A ratio; TV E/é: tricuspid valve E/é ratio; RV FAC: right ventricular fractional area change; GLS RV 4CH: global longitudinal strain right ventricular four chamber view; GLS RV freewall: global longitudinal strain right ventricular freewall; RV SR 4CH: right ventricular strain rate four chamber; RV SR freewall: right ventricular strain rate freewall; peak displ. basal: peak displacement basal; LVEF: left ventricular ejection fraction; GLS LV avg.: global longitudinal strain left ventricular average; MV E/A: mitral valve E/A ratio; MV E/é: mitral valve E/é ratio; Bold writing indicates significance.

influence the preload of the RV, leading to temporary changes in the RV function but the global RV function will return to the baseline value during the first postoperative week, as the RV will adapt to both pre- and afterload changes. Also contrary to our findings, Okada et al. detected RV dysfunction post-thoracic surgery and they speculated that the main cause of RV dysfunction after major pulmonary resection might be the changes in right ventricular afterload. The RV dysfunction was detected by Okada et al. during the first and second PODs, while their measurements of the RV parameters on the third postoperative week demonstrated almost the same values compared to baseline. These findings again strengthen our speculation, that the RV dysfunction, which occurs in the very early postoperative period, diminishes during the first postoperative week, after that the RV adapt to the new pre- and afterload conditions. Major resection of the lung as, for example, in bi-lobectomy, can lead to a reduction of the pulmonary vascular system, which in return leads to reduction of the pulmonary vasculatory cross sections and may lead to PAH and subsequently to right heart failure. Beside changes in the pulmonary vascular system during thoracotomy and anesthesia, an increase in intrathoracic pressures are recorded, which negatively influences the filling pressure of the RV. However, we could not detect any changes neither of systolic RV parameters nor of TV inflow. We speculate that in the acute phase after major lung resection, the RV afterload will increase significantly and the RV function will suffer during the very
early postoperative period trying to adapt to the new afterload. The healthy RV will achieve this adaption during the first 2–3 PODs and at the same time the pulmonary artery pressure and vascular resistance will decrease and normalize again during the first postoperative week.

On the other hand, patients, who have had pericardiotomy during CABG surgery, had a significant reduction of RV longitudinal deformation pattern as demonstrated by reduction of TAPSE and TASV, as well as reduction on global RV function including the transverse pattern as recognized by reduction of RV-FAC, GLS, and SR from global RV as well as GLS and LS from the freewall of the RV. These findings are in discordance with Rainer et al. and Tamborini et al., who found that only the longitudinal deformation patterns of the RV are reduced after cardiac surgery, while the transverse pattern are still preserved. The underlying mechanisms of the reduction of RV function after cardiac surgery remain unclear, but according to our findings and the results from Rainer et al. and Tamborini et al., pericardiotomy seems to play a key role in this phenomenon. It is our hypothesis that the pericardial layers (parietal and visceral layers) support the maintenance of the RV function and especially the longitudinal patterns. Thus, pericardiotomy leads to the loss of the pericardial assistance of the longitudinal deformation of the RV.

RV failure remains a major problem in cardiac surgery, avoiding full pericardiotomy through minimal invasive approach during thoracic surgery or closure of the pericardium after full-sternotomy and pericardiotomy might preserve the RV function and reduce the incidence of postoperative RV failure.

Limitations of the Study

One important limitation of the study that only one acquisition was recorded for all TTE measurements, test–re-test repeatability evaluation was not performed. Echocardiographic studies were performed for clinical questions and not only for the aim of the study, thus leading to non-uniformity of the time-frame in the different surgery cohorts. Our study is limited by the usual shortcomings of a small cohort single-center study. The absence of perioperative and early postoperative echocardiographic analysis and invasive hemodynamic monitoring of the pulmonary circulatory system might be considered a limitation of our study as we only recorded changes in RV function preoperatively and 1 week post-surgery. Owing to the absence of randomization and the partly retrospective nature of our study, our data were subjected to potential bias with regard to patient selection and data acquisition; therefore, caution should be taken when interpreting our results.

Conclusion

Thoracic non-cardiac surgery does not impair global RV function, while patients undergoing cardiac surgery with left open pericardium suffer significant reductions of RV function. Patho-mechanisms of RV impairment after cardiac surgery should be examined prospectively with larger patient cohorts and new cutoffs of TTE parameters for the evaluation of RV function and are subject of an ongoing trial.

Disclosure Statement

All authors have nothing to declare.

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