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ORIGINAL ARTICLE

# Prognostic Importance of Increased Right Ventricular Afterload in Orthotopic Liver Transplantation Recipients With Endstage Cirrhosis

**L.E. Couperus, MD [a], H.W. Vliegen, MD, PhD [a], B.J. Sorgdrager, MD [b], A.C. den Dulk, MD [c], S.L.M.A. Beeres, MD, PhD [a], E.Y. Sarton, MD, PhD [d], J. Dubbeld, MD [e], M.J. Schalij, MD, PhD [a], J.W. Jukema, MD, PhD [a], B. van Hoek, MD, PhD [c], R.W.C. Scherptong, MD, PhD [a*]**

[a]Department of Cardiology, Leiden University Medical Center, Leiden, The Netherlands
[b]Department of Cardiology, Haaglanden Medical Center, The Hague, The Netherlands
[c]Department of Gastroenterology and Hepatology, Leiden University Medical Center, Leiden, The Netherlands
[d]Department of Anaesthesiology, Leiden University Medical Center, Leiden, The Netherlands
[e]Department of Transplant Surgery, Leiden University Medical Center, Leiden, The Netherlands

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**Background** Severely increased right ventricular (RV) afterload is considered a contra-indication for orthotopic liver transplantation (OLT). This study assesses the effects of mildly increased RV afterload on long-term outcome after OLT in relation to RV function.

**Methods** 139 OLT recipients (53 ± 12 years, 76% male) were included. Preoperative RV afterload was assessed invasively or, if not available, echocardiographically and categorised as normal, high-normal (mean pulmonary artery pressure [PAP] 20–25 mmHg or echocardiographic systolic PAP 35–40 mmHg) or mildly elevated (mean PAP 25–35 mmHg or systolic PAP 40–50 mmHg). The association between level of RV afterload, echocardiographic RV function and postoperative outcome was assessed.

**Results** Right ventricular afterload was high-normal in 17% and mildly elevated in 12% of patients. Patients with elevated RV afterload had higher echocardiographic RV dimensions and left ventricular filling pressures. RV functional parameters were within normal range and not associated with RV afterload. Increased RV afterload was associated with a higher incidence of postoperative haemodynamic complications (8%, 17%, and 29% for normal, high-normal and mildly elevated RV afterload, respectively, $p = 0.03$) and worse survival (8-year survival 74%, 41% and 37% respectively, $p = 0.01$). Preoperative RV function was not associated with outcome after OLT.

**Conclusions** Increased RV afterload was associated with increased haemodynamic complications and worse long-term survival in OLT recipients. Right ventricular function in patients with increased RV afterload was within normal range and not associated with postoperative outcome.

**Keywords** Pulmonary artery pressure • Right ventricular function • Orthotopic liver transplantation • Outcome

*Corresponding author at: Leiden University Medical Center, Department of Cardiology, PO Box 9600, 2300 RC Leiden, The Netherlands. Tel.: +31715262020, Fax: +31715266809., Email: r.w.c.scherptong@lumc.nl

# Introduction

Increased right ventricular (RV) afterload is common in patients with endstage liver disease. Right ventricular afterload can increase through multifactorial mechanisms in this population. Firstly, patients with endstage liver disease frequently exhibit a hyperdynamic circulation due to splanchnic vasodilation and the release of humoral factors, which can cause a mild increase in pulmonary pressures [1,2]. Next, the pulmonary vascular resistance can increase through smooth muscle hypertrophy, remodelling and in situ thrombosis resulting in portopulmonary hypertension [3]. This process is thought to be mediated by substances that bypass the liver through collaterals formed in the presence of liver cirrhosis and portal hypertension [3,4]. Furthermore, cirrhotic cardiac involvement can cause left ventricular (LV) dysfunction and elevation of RV afterload due to increased LV diastolic pressure with a subsequent increase in pulmonary wedge pressure [5]. Severely increased RV afterload is an acknowledged risk factor for mortality in patients undergoing orthotopic liver transplantation (OLT) [6,7]. According to current guidelines, OLT is feasible in patients with a mean pulmonary artery pressure (mPAP) <35 mmHg [8,9]. However, the long-term effects of increased RV afterload remain unknown. Right ventricular function is an established determinant of prognosis in various forms of increased RV afterload and could conceptually also be associated with postoperative outcome in OLT recipients with increased RV afterload [10,11]. Therefore, the first objective of the current study was to investigate the long-term course of OLT recipients in relation to preoperative RV afterload. Secondly, in patients with increased RV afterload, the association between preoperative RV function and outcome was assessed.

# Methods

## Study Population

The study population comprised consecutive patients (n = 181) with endstage liver disease who underwent a first liver transplantation at the Leiden University Medical Center between January 2006 and July 2013. All patients underwent extensive preoperative evaluation including echocardiography and, when indicated, invasive pressure measurements [12]. Exclusion criteria were: auxiliary liver transplantation (n = 8); acute liver failure (diagnosed <6 months before transplantation, n = 5); polycystic liver disease (n = 11) or primary liver tumour without pre-existent liver disease (n = 3) as primary indication for transplantation; >2 years duration between assessment of RV afterload and transplantation (n = 11); and insufficient preoperative transthoracic echocardiographic image quality for the current analysis (n = 4). Clinical data were prospectively collected in hospital information systems and retrospectively analysed. In-hospital complications that altered the course of recovery after surgery were registered through case record review. Haemodynamic adverse events, including significant cardiac congestion and haemodynamic important arrhythmia, were noted as haemodynamic complications. All-cause mortality was registered during follow-up through case record review and the national death registry. The study was conducted in accordance with the Helsinki declaration.

## Transthoracic Echocardiography

Preoperative transthoracic echocardiography was performed with the patient in left lateral decubitus and supine position with a commercially available system (Vivid 7 or E9 [General Electric-Vingmed, Horten, Norway]). Images were digitally stored in cine-loop format and analysed using commercially available software (EchoPAC version 112.0.1; General Electric-Vingmed Ultrasound, Horten, Norway). Left ventricular ejection fraction (LVEF) was calculated from the apical four- and two-chamber view using Simpson's biplane technique [13]. End-diastolic left atrial volume was measured in the apical four- and two-chamber view and indexed for body surface area to obtain left atrial volume index (LAVI) [13]. Peak early diastolic mitral inflow velocity (E) was measured on pulsed wave Doppler recordings. Septal and lateral early diastolic mitral annulus velocities were measured on tissue Doppler imaging of the apical four-chamber view and were averaged to acquire E′. The E/E′ ratio was calculated by dividing the peak inflow velocity by the averaged annular velocities [13]. Right ventricular end-diastolic diameter (RVEDD) and tricuspid valve (TV) annulus diameter were measured on the apical RV view [13,14]. The maximum tricuspid regurgitant jet gradient was calculated from continuous wave Doppler using the modified Bernoulli equation in patients with tricuspid regurgitation that allowed reliable assessment of the pressure gradient [15]. Right atrial pressure was estimated as 3, 8 or 15 mmHg, based on the diameter and inspiratory collapse of the inferior caval vein in the subcostal view [13]. Systolic PAP (sPAP) was calculated by summation of the tricuspid regurgitant gradient and right atrial pressure. RV fractional area change (RVFAC) was calculated from RV end-systolic and end-diastolic area tracings in the RV apical view [13]. Tricuspid annular plane systolic excursion (TAPSE) was measured from M-mode recordings of the lateral tricuspid annulus in the RV view [15]. RV longitudinal peak systolic strain (RV LPSS) of the basal, mid-ventricular and apical segments of the RV free wall was measured using speckle-tracking echocardiography and calculated as the average of the three measurements [13].

## Right Heart Catheterisation

During the study period, extensive invasive haemodynamic assessment was not routinely performed. Invasive measurement of hepatic vein pressure was performed in most patients and was extended with assessment of RV haemodynamics when deemed indicated by the investigating radiologist.

## Definition of Elevated RV Afterload

For the purpose of the present study all patients were stratified into categories based on RV afterload. Right ventricular afterload was assessed as part of the screening protocol using

invasive pressure measurement of mPAP (n = 119) or, if not available, using echocardiographic estimation of sPAP (n = 20). High-normal RV afterload was defined as invasively measured mPAP between 20 and 25 mmHg or as echocardiographically estimated sPAP between 35 and 40 mmHg. Mildly elevated RV afterload was defined as mPAP between 25 and 35 mmHg or sPAP between 40 and 50 mmHg [13,16].

## Orthotopic Liver Transplantation

The details of the operation and perioperative care were described previously [17]. Livers were donated after brain death or after circulatory death. Transplantation was standardly performed according to the piggyback technique [18]. Cold ischaemia time was defined as the time between cold flush with preservation fluid in the donor and the removal of the donor liver from ice at the time of the recipient procedure. Recipient warm ischaemic time was defined as the time between liver removal from ice and reperfusion of the donor liver in the recipient. Procedural time refers to the total recipient surgery time.

## Statistical Analysis

Continuous variables are expressed as mean ± standard deviation when normally distributed, or otherwise as median and interquartile range (IQR). Categorical data are presented as frequencies and percentages. Differences in clinical, surgical and echocardiographic characteristics between categories of RV afterload were assessed using the one-way ANOVA, Mann-Whitney U test and Chi-square test, for normally and not-normally distributed continuous data and categorical data, respectively. Kaplan Meier curves for survival after transplantation were plotted for patients with normal, high-normal and mildly elevated RV afterload. A log-rank test was performed to assess differences between these curves. In patients with elevated RV afterload the association between echocardiographic variables and postoperative haemodynamic complications and mortality was assessed using univariate binary logistic regression analysis and multivariate Cox regression analysis, respectively. For this purpose echocardiographic variables were dichotomised based on the median value for the total population to gain a sufficient number of patients in both groups to perform regression analysis. P-values $<0.05$ were considered statistically significant. Statistical analysis was performed using SPSS for Windows (version 23.0, IBM Corp, Armonk, NY, USA).

# Results

## Patient Characteristics

In total, 139 OLT recipients (age at transplantation 53 ± 12 years, 76% male) were included. Table 1 provides baseline characteristics of the population. Most frequent causes of liver disease were alcoholic (49 patients, 35%) and viral (40 patients, 29%). Time between diagnosis of liver disease and OLT was 3.3 years (IQR 1.8–8.6 years). Time between screening assessments and OLT was 203 days (IQR 102–307 days). At the preoperative screening, RV afterload was high-normal in 23 patients (17%) and mildly elevated in 17 patients (12%).

**Table 1 Baseline characteristics of the patient population undergoing OLT.**

| | N = 139 |
|---|---|
| Sex (% men) | 76 |
| Age at transplantation (years) | 53 ± 12 |
| Underlying liver disease: | |
| - Alcoholic (%) | 35 |
| - Viral (%) | 29 |
| - Primary sclerosing cholangitis (%) | 12 |
| - Cryptogenic (%) | 5 |
| - Metabolic (%) | 5 |
| - Primary biliary cirrhosis (%) | 5 |
| - Non-alcoholic steatohepatitis (%) | 4 |
| - Auto-immune (%) | 3 |
| - Other (%) | 4 |
| High-normal RV afterload (%) | 17 |
| Mildly elevated RV afterload (%) | 12 |

Abbreviations: OLT, orthotopic liver transplantation; RV, right ventricular.

Table 2 presents patient characteristics stratified per category of RV afterload. Patients with normal, high-normal and mildly elevated RV afterload did not differ significantly in sex, age at transplantation, duration of liver disease, model for endstage liver disease score or hepatic venous pressure gradient. Preoperatively a transjugular intrahepatic portosystemic shunt (TIPS) procedure was performed in four patients (24%) with mildly elevated RV afterload versus seven patients (7%) with normal and 0 patients (0%) with high-normal RV afterload ($p = 0.02$).

## Preoperative Echocardiographic Assessment

Preoperative echocardiography was performed in all patients as shown in Figure 1. Left ventricular ejection fraction and LAVI were not related to the level of RV afterload (Figure 1 AB). Patients with increased RV afterload had higher left ventricular filling pressures on echocardiography (E/E′ ratio 10 ± 3, 13 ± 5 and 12 ± 4 [$p < 0.01$] in patients with normal, high-normal and mildly elevated RV afterload, respectively; Figure 1 C). Furthermore, patients with increased RV afterload had higher RV dimensions (RVEDD 36 ± 4 mm, 37 ± 4 mm and 39 ± 5 mm [$p < 0.01$] and TV annulus 29 ± 5 mm, 29 ± 4 mm and 32 ± 4 mm [$p = 0.01$] in patients with normal, high-normal and mildly elevated RV afterload, respectively; Figure 1 DE). Right ventricular function measured as RVFAC, TAPSE and RV LPSS was within normal range and not associated with the level of RV afterload (Figure 1 FGH) [13,16].

**Table 2 Clinical, surgical and postoperative characteristics of patients undergoing OLT with preoperative normal, high-normal and mildly elevated RV afterload.**

| | Normal RV afterload N = 99 | High-normal RV afterload N = 23 | Mildly elevated RV afterload N = 17 | P |
|---|---|---|---|---|
| **Clinical characteristics** | | | | |
| Age at transplantation (years) | 52 ± 12 | 57 ± 11 | 52 ± 12 | 0.20 |
| Sex (% men) | 80 | 61 | 71 | 0.14 |
| Duration liver disease (years) | 3.1 (1.8-6.7) | 5.5 (2.0-15.3) | 3.4 (1.4-11.0) | 0.31 |
| Alcoholic aetiology of liver disease (%) | 35 | 26 | 47 | 0.39 |
| MELD score | 21 (15-24) | 21 (16-28) | 23 (19-24) | 0.54 |
| Preoperative TIPS procedure (%) | 7 | 0 | 24 | **0.02** |
| **Invasive pressure measurement** | | | | |
| Hepatic venous pressure gradient (mmHg) | 22 ± 9 | 19 ± 10 | 18 ± 11 | 0.20 |
| Mean PAP (mmHg) | 14 ± 3 | 22 ± 1 | 28 ± 2 | **<0.01** |
| **Surgical characteristics** | | | | |
| Donation after cardiac death (%) | 34 | 22 | 18 | 0.24 |
| Cold ischaemic time (mins) | 545 ± 135 | 561 ± 153 | 564 ± 125 | 0.80 |
| Warm ischaemic time (mins) | 35 ± 9 | 37 ± 8 | 37 ± 11 | 0.60 |
| Procedural time (mins) | 348 ± 127 | 365 ± 101 | 358 ± 102 | 0.84 |
| **Postoperative follow-up** | | | | |
| Postoperative complications (%) | 50 | 61 | 65 | 0.37 |
| Postoperative haemodynamic complications (%) | 8 | 17 | 29 | **0.03** |
| Duration hospital stay (days) | 12 (10-23) | 16 (10-34) | 13 (10-36) | 0.08 |
| In-hospital mortality (%) | 5 | 13 | 12 | 0.30 |

Abbreviations: OLT, orthotopic liver transplantation; RV, right ventricular; MELD, Model for Endstage Liver Disease.
Bold values signify statistical significance.

## Surgical Data and Postoperative Outcome

Orthotopic liver transplantation with a liver donated after circulatory death was performed in 42 patients (30%). Mean cold and recipient warm ischaemic time and procedural time were 550 ± 136 minutes, 36 ± 9 minutes and 352 ± 120 minutes, respectively. The incidence of postoperative complications was 53%. In particular, haemodynamic complications developed in 12% of patients and consisted of postoperative haemodynamic instability with prolonged inotrope dependence (seven patients), development of respiratory insufficiency caused by cardiac congestion (six patients), arrhythmia with haemodynamic instability (three patients) and endocarditis with cardiac congestion (one patient). Median duration of hospital stay was 13 days (IQR 10–24 days) and in-hospital mortality was 7%. In-hospital mortality was related to circulatory failure in 8 out of 10 patients (80%) and comprised multi-organ failure in 5 patients and sudden cardiac death in 3 patients. One in-hospital death was transplant related and the cause of death was unknown in one patient. Median follow-up time after OLT was 4.8 years (IQR 2.8–6.6 years). Overall, 37 patients (27%) died during follow-up of which 13 patients (35%) died from circulatory failure; multi-organ failure in eight patients, sudden cardiac death in four patients and heart failure in one patient. Other causes of death were carcinoma in 11 patients, transplant related in four patients and unknown in nine patients. Patient survival 1, 3, 5 and 8 years after OLT was 87, 81, 74 and 62%, respectively.

## Association Between RV Afterload and Outcome

Haemodynamic complications occurred more frequently in patients with increased RV afterload (8% in patients with normal RV afterload, 17% in patients with high-normal RV afterload and 29% in patients with mildly elevated RV afterload, p = 0.03). Increased RV afterload was not reflected in a higher incidence of in-hospital mortality. However, long-term survival after OLT was significantly impaired for patients with increased RV afterload, as presented in Figure 2. The 8-year survival rate was 74% for patients with normal RV afterload, 41% for patients with high-normal RV afterload and 37% for patients with mildly elevated RV afterload (p = 0.01).

## Association Between RV Function and Outcome in Patients With Elevated RV Afterload

In patients with increased RV afterload, the implications of echocardiographic parameters on postoperative outcome were assessed. Echocardiographic parameters were dichotomised

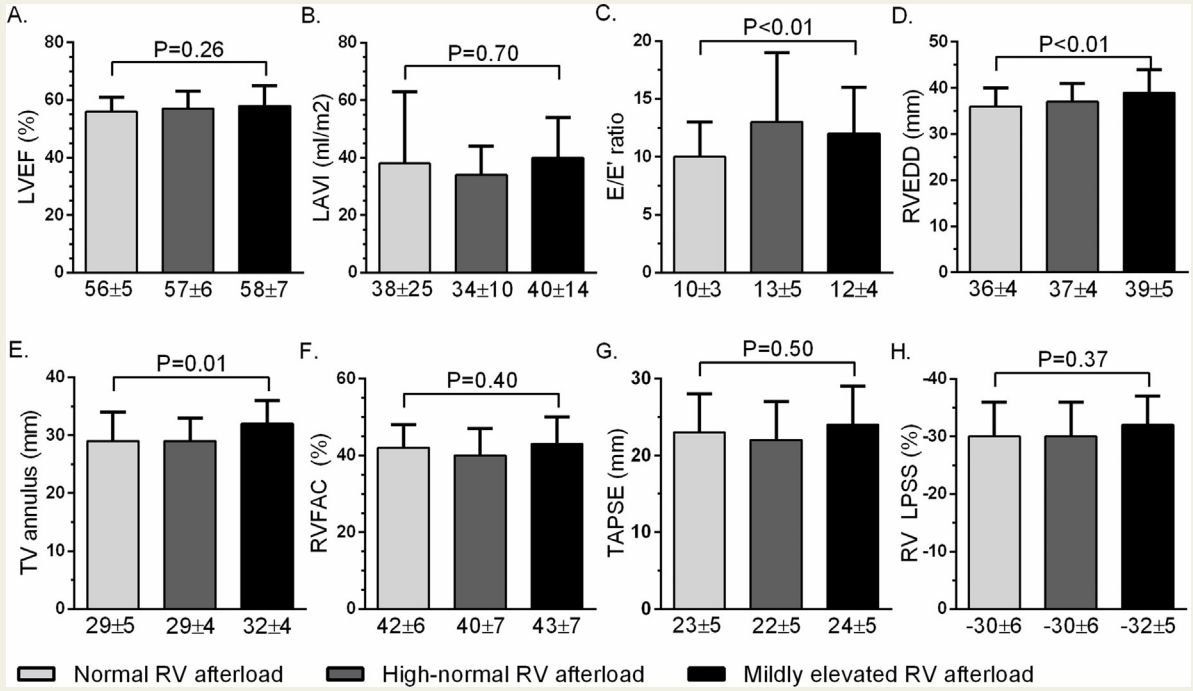


**Figure 1** Echocardiographic characteristics of patients undergoing OLT with preoperative normal, high-normal and mildly elevated RV afterload.
Abbreviations: OLT, orthotopic liver transplantation; RV, right ventricular; LVEF, left ventricular ejection fraction; LAVI, left atrial volume index; E/E', peak early diastolic mitral inflow velocity/early diastolic mitral annulus velocity; RVEDD, right ventricular end-diastolic diameter; TV, tricuspid valve; RVFAC, right ventricular fractional area change; TAPSE, tricuspid annular plane systolic excursion; RV LPSS, right ventricular longitudinal peak systolic strain.

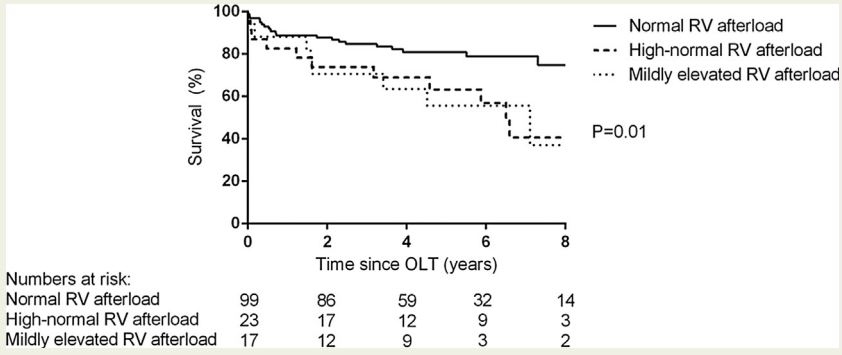


**Figure 2** Kaplan Meier curves for survival after OLT in patients with preoperative normal, high-normal and mildly elevated RV afterload.
Abbreviations: OLT, orthotopic liver transplantation; RV, right ventricular.

based on the median value for the total population of patients with increased RV afterload. As presented in Table 3, multivariate binary logistic regression analysis and univariate Cox regression analysis revealed that RV echocardiographic parameters in patients with increased RV afterload were neither related to the incidence of postoperative haemodynamic complications nor to mortality after OLT after adjusting for age and sex.

## Discussion

The main findings of the present study were: 1) patients with increased RV afterload had significantly more postoperative haemodynamic complications and reduced long-term survival after OLT; and 2) preoperative echocardiographic evaluation of RV function in these patients was within normal range and not associated with postoperative outcome.

**Table 3 Odds ratios for haemodynamic complications and hazard ratios for mortality after OLT in patients with preoperative high-normal and mildly elevated RV afterload.**

| | Haemodynamic complications | | | | Mortality | | |
|---|---|---|---|---|---|---|---|
| | N | OR | 95%CI | P | HR | 95%CI | P |
| LVEF <57% | 19 | 0.85 | 0.19-3.79 | 0.84 | 2.11 | 0.84-5.30 | 0.11 |
| LAVI >34 ml/m $^2$ | 20 | 12.67 | 1.40-114.42 | **0.02** | 2.30 | 0.90-5.86 | 0.08 |
| E/E' ratio >11 | 22 | 0.53 | 0.12-2.40 | 0.41 | 0.86 | 0.34-2.13 | 0.74 |
| RVEDD >38 mm | 16 | 1.42 | 0.30-6.81 | 0.66 | 2.32 | 0.86-6.27 | 0.10 |
| TV annulus >31 mm | 17 | 0.64 | 0.13-3.20 | 0.59 | 1.12 | 0.41-3.06 | 0.82 |
| RVFAC <40% | 21 | 0.66 | 0.15-2.93 | 0.58 | 0.44 | 0.17-1.13 | 0.09 |
| TAPSE <23 mm | 18 | 0.97 | 0.22-4.32 | 0.97 | 1.22 | 0.49-3.00 | 0.67 |
| RV LPSS >-30% | 17 | 0.60 | 0.12-3.01 | 0.53 | 0.80 | 0.30-2.11 | 0.65 |

Abbreviations: HD, haemodynamic, OLT, orthotopic liver transplantation; RV, right ventricular; LVEF, left ventricular ejection fraction; LAVI, left atrial volume index; TV, tricuspid valve; RVEDD, right ventricular end-diastolic diameter; sPAP, systolic pulmonary artery pressure; RVFAC, right ventricular fractional area change; TAPSE, tricuspid annular plane systolic excursion; RV LPSS, right ventricular longitudinal peak systolic strain.
Bold values signify statistical significance.

## Aetiology of Increased RV Afterload in Endstage Liver Disease

Right ventricular afterload can increase through multifactorial mechanisms in patients with endstage liver disease. Endstage liver disease is characterised by dynamic and structural vascular changes, including splanchnic vasodilatation, splanchnic collateral formation and increased blood flow [1,20]. This increase in blood flow can lead to a hyperdynamic circulation and a mild increase in pulmonary pressures [1,2]. Next, formation of splanchnic collaterals is thought to enable substances to bypass the liver and cause smooth muscle hypertrophy, remodelling and in situ thrombosis of the pulmonary vasculature. Through this pathway pulmonary vascular resistance can increase which can lead to portopulmonary hypertension [3,4]. Moreover, haemodynamic changes in endstage liver disease can lead to cirrhotic cardiomyopathy with LV systolic and diastolic dysfunction. The resultant increase in LV filling pressures leads to elevation of the pulmonary capillary wedge pressure and thus elevation of pulmonary pressure and RV afterload [5,19].

The concept that multifactorial mechanisms can be involved in the development of RV overload is supported by the present data. Firstly, patients with higher RV afterload had a higher frequency of prior TIPS insertion. It is hypothesised that TIPS insertion may be a risk factor for an increase in pulmonary vascular resistance as well as development of cirrhotic cardiomyopathy, as it induces shunting of vasoactive and pro-inflammatory substances to the pulmonary circulation [20,21]. Next, RV dimensions in the present study were slightly increased (RVEDD 36 ± 4 mm in patients with normal RV afterload, normal reference range 33 ± 4 mm) and were higher in patients with increased RV afterload [13]. Kia and colleagues also found a high-normal RV diameter (40 ± 7 mm) in a population of endstage liver disease patients prior to transplantation, but in this study no stratification for RV afterload was performed [21]. An explanation for the high RV dimensions may be the increased blood flow (hyperdynamic circulation) that increases RV end-diastolic volume. Furthermore, a higher E/E' ratio, indicating higher LV filling pressures, was observed in patients with elevated RV afterload. Higher LV filling pressures can be the result of LV systolic and diastolic dysfunction. This finding is consistent with the studies by Benjaminov and Yassen and colleagues, who found higher pulmonary capillary wedge pressures, as marker of LV end-diastolic pressures, in liver disease patients with pulmonary hypertension as compared to patients without pulmonary hypertension [22,23]. The aetiology of increased RV afterload in OLT recipients in the present study therefore appears multifactorial, involving a combination of the above described mechanisms.

## RV Overload and Outcome After OLT

So far, literature on RV overload in OLT recipients mostly concerns patients with portopulmonary hypertension [3]. Portopulmonary hypertension is a specific disease entity and, once severe, an established risk factor for poor outcome in OLT recipients [6,24]. However, the present study already demonstrated an association between high normal and mildly increased RV afterload and an increased incidence of haemodynamic complications and worse long-term survival after OLT. An initial increase in RV afterload can be a first sign of haemodynamic impairment caused by multifactorial pathways and might already have a negative impact on outcome after OLT. A study by Bozbas and colleagues investigated postoperative complications in OLT recipients [25]. In this study the presence of portopulmonary hypertension was related to the incidence of respiratory complications, such as pleural effusion and respiratory failure. The development of these postoperative complications could have been enhanced by pre-existent RV overload and these findings therefore appear consistent with the results of the present study.

## Relevance of Preoperative RV Function in OLT Recipients With Elevated RV Afterload

In this study, preoperative RV function on echocardiography was within normal range. This is consistent with a previous study by Kia and colleagues, who assessed patients before liver transplantation and measured values of RVFAC and TAPSE within the reference standard of the American Society of Echocardiography guidelines [21]. Right ventricular function is a major determinant of survival in various populations with elevated RV afterload [10,11]. One of the objectives of the present study was, therefore, to investigate the association between echocardiographic characteristics and outcome after OLT in patients with elevated RV afterload. In the present study, however, preoperative RV function was not associated with outcome after OLT in patients with elevated RV afterload. Limited data are available on the association between preoperative RV parameters and outcome in OLT recipients. Kia and colleagues found that mild or worse tricuspid regurgitation was independently predictive of mortality and graft failure in OLT recipients [21]. However, it has to be noted that the severity of tricuspid regurgitation is in general correlated with the degree of RV afterload.

## Limitations

Some limitations should be acknowledged. The study comprised retrospective data from a single centre. Median time between screening assessments and OLT was 203 days (IQR 102–307 days) and RV afterload could have changed in the meantime. Previous studies showed a progression in RV afterload from screening to surgery, which could have resulted in underestimation of RV afterload at the time of OLT. Colle and colleagues reported a study of 165 OLT recipients in which three patients had developed portopulmonary hypertension between screening and surgery [26]. Chiva and colleagues detected a mean increase of 30% in mPAP between screening and OLT in a subgroup of cirrhosis patients undergoing OLT with available measurements at both time points [20]. Furthermore, invasive measurement of cardiac output and LV end-diastolic pressure was only performed when deemed indicated by the investigating radiologist. Therefore, these measurements could not be taken into account in the present analysis. Future research is necessary and should include structural invasive haemodynamic assessment to further explore the contribution of increased cardiac output, left ventricular pressures and increased transpulmonary gradient on the development of right ventricular overload and on outcome in this population.

## Clinical Implications

The present findings indicate that patients with mildly elevated RV afterload should be monitored closely for potential progression of RV afterload and development of perioperative complications in the setting of OLT. Moreover, in line with previous studies, the present data emphasise the relevance and implications of cardiac screening in OLT candidates [27]. Right heart catheterisation remains the gold standard for assessment of RV afterload and should therefore be strongly considered in all OLT candidates [28]. Follow-up of RV pressures after OLT may be indicated to facilitate timely diagnosis of RV overload. In patients with RV overload the aetiology of the increase in afterload needs to be determined to deliver tailored treatment. Future research is necessary to gain insight into the long-term course of RV pressure and function after OLT.

# Conclusion

Preoperative high-normal and mildly elevated RV afterload was associated with increased incidence of haemodynamic complications and worse long-term survival in OLT recipients. Preoperative echocardiographic RV function in patients with elevated RV afterload was within normal range and not associated with postoperative outcome.

# Disclosures

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