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Preoperative Pulmonary Hypertension and In-Hospital Outcomes After Pulmonary Valve Replacement In Adults with Congenital Heart Disease: A National Inpatient Sample Analysis

Soujanya Bogarapu
University of Illinois College of Medicine at Peoria
Joshua Benjamin Merrill
University of Illinois College of Medicine at Peoria
Ryan Subah Yeleti
Dunlap High School
Marc Douglas Knepp
University of Illinois College of Medicine at Peoria
Subhrajit Lahiri
subrajit.lahiri@ouhealth.com
University of Oklahoma

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"Preoperative Pulmonary Hypertension and In-Hospital Outcomes After Pulmonary Valve Replacement In Adults with Congenital Heart Disease: A National Inpatient Sample Analysis"

Bogarapu S, MD[1]; Merrill J[2]; Yeleti RS[3]; Knepp MD, MD[1]; Lahiri S, MD[4]

Institutional Affiliations:

[1]Division of Pediatrics Cardiology, Department of Pediatrics, University of Illinois College of Medicine at Peoria, Peoria, IL, USA

This author take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation

[2]University of Illinois College of Medicine at Peoria, Peoria, IL, USA.

This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation

[3]Dunlap High School, Peoria, IL, USA

This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation

[4]Division of Pediatric Cardiology, Department of Pediatrics, University of Oklahoma, Oklahoma, USA.

This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation

Corresponding Author:
Subhrajit Lahiri, MD
Department of Pediatrics, Division of Pediatric Cardiology
University of Oklahoma
Oklahoma
Ph: 309-655-3453
Fax: 309-655-9242
Email: subhrajit.lahiri@ouhealth.com

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Structured Abstract:

Background: Pulmonary valve replacement (PVR) is a cornerstone intervention for adults with congenital heart disease (ACHD), but data on how preoperative pulmonary hypertension (PH) affects outcomes are limited.

Methods: We examined in-hospital outcomes among ACHD patients with and without PH undergoing PVR using National Inpatient Sample (2018–2020). Outcomes were in-hospital mortality and length of stay (LOS), assessed with multivariable logistic, Firth-penalized, and log-linear regression adjusting for age, sex, and comorbidities.

Results: Among 1,533 adults undergoing PVR (mean age 27±19 years, 40.5% female), 138 (9%) had PH and 502 (32%) had tetralogy of Fallot. Overall mortality was low (1.3%). PH patients were older (39±22 vs 26±18 years, $p<0.001$), more often female (53% vs 40%, $p=0.004$), and had more COPD (8.7% vs 1.3%, $p<0.001$) and emphysema (2.2% vs 0.2%, $p=0.005$). Unadjusted mortality was four-fold higher with PH (OR 4.48, 95% CI 1.57-11.38), but attenuated after adjustment (Firth OR 1.88, 95% CI 0.54-5.68); age emerged as the dominant predictor (OR 1.04/year, optimal threshold of 45.5 years). Median LOS was 8 (IQR 4–14) vs 4 days (IQR 2–7), and PH was independently associated with a 56% longer hospitalization (95% CI 35-82%); emphysema had the largest relative effect (+143%).

Conclusion: A coded PH diagnosis identifies a higher-risk PVR phenotype, older, more often female, with pulmonary comorbidity, and roughly 50% longer hospitalization despite no clear increase in adjusted mortality. With age dominant beyond 45 years, PH is a marker of complexity warranting earlier intervention and structured care in specialized ACHD-PH programs.

Key Words: Pulmonary hypertension, pulmonary valve replacement, outcomes, length of stay

Introduction:

Pulmonary valve replacement (PVR) is a cornerstone intervention for adults with congenital heart disease grappling with pulmonary valve dysfunction, particularly when complicated by right ventricular (RV) dilation, progressive RV dysfunction, exercise intolerance, and potential left ventricular (LV) impairment[1-5]. Although most patients requiring PVR have normal pulmonary artery (PA) pressures, there is a notable subset that may have preoperative pulmonary hypertension (PH). This complication can stem from a variety of reasons, such as late diagnosis, delays in initial interventions, chronic exposure to systemic-to-pulmonary artery shunts, residual shunt lesions, or concomitant non-cardiac morbidities that further challenge an already strained cardiovascular physiology[6-7]. The presence of preoperative PH can influence surgical risk stratification, impact perioperative decision-making, and alter postoperative recovery trajectories[8]. Despite its critical clinical implications, there is little current literature about how preoperative PH affects outcomes after PVR, and this important question underscores the need for further investigation.

Our study aims to address this knowledge gap by comparing outcomes in the adult congenital heart disease (ACHD) patients with and without PH undergoing PVR. Using a large national database, we examined how PH influences hospital stays and survival in this patient group. Our goal is to better understand these outcomes and provide insights to improve patient care.

Methods:

Data Source and Study Design

We conducted a retrospective cohort analysis utilizing the National Inpatient Sample (NIS) database from 2018 to 2020. The NIS, part of the AHRQ-sponsored Healthcare Cost and Utilization Project, is the largest publicly accessible all-payer inpatient database in the U.S, capturing roughly 20% of hospital discharges with sampling weights that allow valid national estimates. It includes data on inpatient

utilization, costs, quality, and clinical outcomes, with over 7 million adult admissions (≥18 years) recorded annually.

Study Population and Case Identification

Our study cohort comprised adult patients (≥18 years) who underwent pulmonary valve replacement during the study period. Pulmonary valve replacement procedures were identified using ICD-10-PCS codes 02RH0KZ (replacement of pulmonary valve with synthetic substitute, open approach), 02RH3KZ (percutaneous approach), and 02RH4KZ (percutaneous endoscopic approach). Pre-existing pulmonary hypertension was defined using ICD-10-CM diagnosis codes I27.0, I27.2, I27.20, and I27.21, and hypertension, diabetes mellitus, chronic obstructive pulmonary disease, emphysema, obesity, and asthma were identified using standard ICD-10-CM code families (e.g., I10, E11.xx, J44.x, J43.9, E66.x, J45.x); the complete list of codes is provided in Supplemental Table S1.

Outcomes Assessment and Statistical Analysis

The primary endpoint was in-hospital mortality, defined as death occurring during the index hospitalization for PVR. Our secondary endpoint was length of stay (LOS), measured from admission through discharge. Patients who died during hospitalization were excluded from LOS calculation to prevent bias from artificially shortened stays due to in-hospital mortality.

Comparative analyses of patient characteristics between PH and non-PH cohorts were performed using chi-square tests for categorical variables and t-tests for continuous variables. Data normality was assessed using multiple methods, including skewness and kurtosis statistics, visual inspection of histograms and Q-Q plots, and the Shapiro-Wilk test. Length of stay exhibited significant right skewness (skewness = 7.8, kurtosis = 90.8) and is therefore reported as median with interquartile range [IQR]. Age demonstrated approximately normal distribution (skewness = 0.74) and is reported as mean ± standard deviation (s).

Mortality was compared between groups using logistic regression, and LOS using linear regression. All models were adjusted for clinically relevant comorbidities: obesity, diabetes mellitus, hypertension, asthma, chronic obstructive pulmonary disease (COPD) and emphysema. Chronic kidney disease was described but not modeled given its low prevalence and to preserve a stable events-per-variable ratio in mortality analysis. PH severity, PH-directed therapies, and surgical versus transcatheter PVR cannot be reliably identified from administrative codes and were not modeled.

All analyses incorporated survey-specified statistical techniques to account for the complex sampling design inherent to the NIS database. Statistical significance was defined as $p < 0.05$, and all analyses were performed using R version (4.5.1).

Results:

From the nearly 21 million weighted discharges in the NIS database during the study period, we identified 1,533 patients who underwent PVR. Of these patients, 138 (9%) had concurrent PH while 1,395 (91%) did not have pulmonary hypertension.

Table 1 presents the demographic and clinical characteristics comparing patients with and without PH. The overall cohort had a mean age of 27±19 years, reflecting a predominantly young adult population with considerable age distribution. Patients with PH represented a smaller proportion of the cohort (138/1395; 9%) but exhibited a distinctly higher-risk phenotype. They were older (39±22 years vs 26±18 years, $p<0.001$) and had longer hospitalizations (median 8 [IQR 4-14] vs 4 [IQR 2-7] days, $p<0.001$). Sex distribution differed modestly, with a lower proportion of males in the PH group (47% vs 60%, $p=0.004$).

Pulmonary comorbidities were notably enriched among PH patients, who had higher rates of COPD (8.7% vs 1.3%, $p<0.001$) and emphysema (2.2% vs 0.2%, $p=0.005$). Diabetes trended higher in PH patients (9.4% vs 5.2%, $p=0.06$), while comorbidities, including hypertension, and asthma, showed no significant difference between groups.

Tetralogy of Fallot (TOF) emerged as the most common underlying heart defect in our cohort, seen in 502 patients (32%). This finding highlights TOF as a leading CHD requiring PVR intervention.

Mortality Outcomes

The observed in-hospital mortality was low overall, occurring in just 20 patients (1.3%), reflecting the general safety of PVR in this population. However, the mortality rates were dramatically different between groups, with PH patients experiencing a four-fold in-patient mortality rate (4.3%) compared to non-PH patients (1.0%, p=0.004).

In unadjusted analysis, PH was associated with significantly increased odds of mortality (OR 4.48, 95% CI 1.57-11.38, p=0.002). However, after adjusting for age, sex, and comorbidities using multivariable logistic regression, the association between PH and mortality was attenuated and no longer statistically significant (OR 1.84, 95% CI 0.51-5.72, p=0.32). Given the low event count, we additionally fitted a Firth-penalized logistic regression as our preferred adjusted estimate, which yielded similar results (OR 1.88, 95% CI 0.54-5.68). In both adjusted models, increasing age emerged as the primary independent predictor of mortality (OR 1.04 per year, 95% CI 1.02-1.07, p<0.01; Figure 1), with mortality risk concentrated in patients over 45 years of age. COPD showed a trend towards increased mortality risk (OR 4.11, 1.2-15.5, p=0.02), though other comorbidities were not significant predictors, likely due to limited events. Age demonstrated good discriminative ability for in-hospital mortality, with an optimal ROC-derived cutoff of 45.5 years (sensitivity 0.65, specificity 0.82; Figure 2)

Given the low total number of deaths (n=20), we conducted a post-hoc power analysis which revealed 69% power to detect the observed mortality difference (4.3% vs 1.1%, p<0.05). While this falls below the conventional 80% threshold, it suggests that non-significant adjusted results should be interpreted cautiously, as they may reflect statistical power limitations rather than absence of clinical effects.

Length of Stay

There was significant difference between LOS between the two groups. Patients with PH had significantly longer hospitalization compared to those without PH. The median LOS was 8 days [IQR 4-14] for PH patients versus 4 days [IQR 2-7] for non-PH patients (p=0.001; Figure 3a). This represents a Hodges-Lehmann median difference of 3.0 days (95% CI 2.0-4.0, p=0.001). The overall cohort median LOS was 4 days, although the distribution was highly right-skewed, with some patients experiencing substantially prolonged hospitalizations.

In multivariable linear regression of log-transformed LOS among survivors, PH remained independently associated with a 56% longer hospitalization (95% CI 35-82%, p<0.002) after adjusting for age, sex, and comorbidities. Among comorbidities, only emphysema significantly increased LOS (+143%, 95% CI 19-339%, p=0.01; Figure 3b), while obesity, hypertension, diabetes, COPD, and asthma were not significant predictors of hospital duration.

Discussion:

This National cohort study represents one of the largest contemporary analyses examining the impact of PH on perioperative outcomes following PVR in ACHD. Our findings clearly demonstrate that PH reshapes the clinical profile of patients undergoing PVR, identifying a distinct higher-risk subset characterized by older age, female predominance, and a greater burden of pulmonary comorbidities. Most notably, PH was associated with a significantly prolonged hospital LOS. Despite these baseline differences, in-hospital mortality remained low and comparable between groups. This underscores that while PVR is generally safe, the presence of PH confers a fundamentally different perioperative risk profile that warrants deliberate recognition and specialized management.

Pulmonary hypertension as a high-risk phenotype

Pulmonary hypertension affects approximately 5–10% of adults with congenital heart disease and remains an established determinant of morbidity and mortality[10, 11]. In the present cohort, 9% of patients undergoing PVR had documented PH, consistent with prior reports. Despite improvements in targeted

therapies, contemporary PH-CHD cohorts after surgical correction continue to demonstrate 5-year survival of approximately 90%, underscoring the persistent clinical burden of this condition[12]. Rather than a straightforward volume or pressure unloading procedure, PVR in this setting addresses a more complex pathophysiology with impaired RV–pulmonary vascular coupling and limited hemodynamic reserve. In our analysis, PH was independently associated with an approximately 50% increase in LOS, even after adjustment for age and comorbidities, consistent with prior work identifying RV dysfunction as a central driver of adverse outcomes in PH. Postoperative RV failure after cardiac surgery has been associated with a 3.6-fold higher risk of a composite outcome including death, stroke, reintubation, and prolonged intensive care unit (ICU) stay[13]. Multidisciplinary perioperative PH pathways and the use of pulmonary vasodilators such as inhaled nitric oxide, phosphodiesterase-5 inhibitors, prostacyclin analogues, and endothelin receptor antagonists are recommended to reduce pulmonary vascular resistance and stabilize RV function. The clinical profile of PH in this ACHD cohort therefore aligns with its established role as a marker of advanced cardiopulmonary disease and heightened perioperative risk.

Hospital Length of Stay and Resource Utilization

The significantly prolonged hospitalization among PH patients stands out as one of the most clinically important findings. An adjusted 56% increase in LOS underscores the substantial resource utilization and complexity associated with PH in this setting. Similar patterns have been described elsewhere: Desai et al. reported increased LOS among PH patients undergoing elective non-cardiac surgery, while Melby et al. and Vincens et al. observed comparably prolonged hospitalizations in patients with PH undergoing aortic valve replacement for aortic stenosis and mitral surgery for severe mitral stenosis, respectively[14-16]. Prolonged hospitalization in our cohort likely reflects the interplay of complex perioperative hemodynamic management, a higher incidence of RV dysfunction, increased susceptibility to respiratory and hospital-acquired complications, and the need for tailored pharmacologic support. These extended stays translate into greater healthcare resource consumption and may negatively affect quality of life, delay return to normal activities, and influence longer-term outcomes[17]. The positively skewed

distribution of LOS further suggests that a subset of patients, likely those with more severe PH or additional comorbidities, experience particularly protracted recovery, underscoring the importance of identifying predictors of extended hospitalization and designing targeted care pathways for high-risk patients.

Clinical Risk Stratification: Age as the Critical Factor

Perhaps the most compelling finding of our study is the identification of age as the dominant determinant of in-hospital mortality following PVR, overshadowing even the presence of PH itself. PH was associated with an approximately four-fold higher unadjusted in-hospital mortality (4.3% vs 1.0%), but this effect was markedly attenuated after multivariable adjustment, indicating that advancing age largely drove this association. The age–mortality relationship was sufficiently pronounced that patients older than 45 years accounted for a disproportionate share of deaths, irrespective of PH status.

The identification of 45.5 years as the optimal mortality threshold (sensitivity 0.65, specificity 0.82) has important implications for timing of intervention in ACHD. Although current recommendations favor earlier PVR to prevent irreversible RV remodeling, many patients still undergo surgery late in the disease course. Prior work has demonstrated diminished RV reverse remodeling and a 5.6-fold higher 10-year mortality when PVR is performed after age 35 years in repaired tetralogy of Fallot[18]; our data extend these observations to a broader ACHD population, reinforcing that advancing age, particularly beyond the mid-40s, marks a critical inflection point in perioperative risk.

This powerful age effect likely reflects cumulative myocardial injury from chronic volume or pressure overload, resulting in fibrosis, impaired contractile reserve, and reduced capacity for recovery after surgical correction. The substantially older age of patients with PH further suggests delayed recognition or evolution to a more advanced disease phenotype. Together, these findings emphasize that timing of intervention, rather than PH status alone, may represent the more modifiable determinant of mortality risk in ACHD patients undergoing PVR.

Equal Mortality Despite Disparate Risk Profiles

The most unexpected finding was the similar in-hospital mortality between PH and non-PH patients, given how consistently PH worsens outcomes in other cardiac surgical settings[19-22], despite clear differences in age, comorbidity burden, and LOS. A few explanations seem likely. First, our overall mortality was low (1.3%), suggesting that PVR has become a relatively safe operation in experienced centers. Second, surgical candidacy for PH is also highly selective: patients with end-stage RV failure or severe, irreversible pulmonary vascular disease are usually steered toward medical or transplant pathways, so those who do reach the operating room may represent a relatively "healthy" subset with preserved reserve. Third, once PH is recognized, teams often adopt a more aggressive perioperative strategy, with invasive monitoring, early use or escalation of PH-directed therapies, lower thresholds for mechanical support, and longer ICU stays. Finally, our endpoint was in-hospital mortality, which may miss deaths after discharge or RV decompensation, where PH could still have a greater impact. Taken together, PH did not translate into higher in-hospital mortality in this carefully selected cohort, but it remained a marker of a more complicated perioperative course with important consequences for patients and resource use.

Comorbidity burden with disproportionate impact of emphysema

Emphysema was associated with the largest relative increase in LOS, in our adjusted analysis (>150%), though confidence intervals were wide given the small number of affected patients, and COPD showed a similar trend. Traditional cardiovascular risk factors, including diabetes, hypertension, and obesity, had only modest effects and were not consistent predictors. This pattern is clinically plausible, as chronic lung disease is known to increase postoperative respiratory complications and prolong recovery after cardiac surgery[23, 24]. Emphysema-related PH reflects reduced pulmonary vascular reserve which, when added to the hemodynamic burden of CHD, may further limit RV adaptation in the perioperative period. Pulmonary comorbidity therefore carries disproportionate prognostic weight in this population and deserves careful attention during preoperative assessment and risk counseling.

Sex Differences and Referral Patterns

The striking female predominance in the PH cohort (53% vs 40%) warrants attention, particularly given the overall male predominance in PVR procedures. This mirrors the recognized female predominance in PH and PH-CHD populations[25, 26]. Across cardiac surgical populations, women have consistently higher short-term mortality than men, with meta-analyses reporting 1.3–1.8-fold increased odds of 30-day mortality after CABG and valve surgery[27, 28]. Proposed mechanisms include smaller body and vessel size, lower baseline hemoglobin with greater vulnerability to intraoperative anemia, and potential underuse of arterial grafts or guideline-directed therapies[29, 30]. In the current study, however, in-hospital mortality did not differ by sex, suggesting that in an experienced ACHD center with structured PH management pathways, sex-based disparities may be attenuated. Still, the combination of female sex, PH, and advanced age delineates a subgroup at heightened risk for prolonged LOS and resource use, warranting tailored preoperative counseling and perioperative strategies.

Clinical Implications and Future Directions

Our findings have several important clinical implications. First, our data argues against viewing PH as an absolute contraindication to PVR. Instead, PH should be recognized as a high-risk phenotype that requires: (1) earlier identification and referral before advanced age and irreversible RV damage ensue; (2) rigorous preoperative characterization of pulmonary vascular disease and RV function using invasive hemodynamics and advanced imaging; (3) aggressive optimization of PH and pulmonary comorbidities, particularly emphysema; and (4) management at high-volume ACHD centers with multidisciplinary teams capable of delivering complex perioperative PH care. The disproportionate impact of emphysema suggests that structured pulmonary rehabilitation, smoking cessation, and targeted bronchodilator therapy are likely to yield outsized benefits in this group. The growing availability of transcatheter PVR may further reduce physiological stress in high-risk PH patients, although comparative effectiveness studies in this specific subgroup are needed.

A structured perioperative care pathway for these patients can be conceptualized in five phases. The *preoperative* phase should include right heart catheterization with assessment of pulmonary vascular resistance and vasoreactivity, cardiac magnetic resonance imaging for right ventricular volumes, ejection fraction, and pulmonary regurgitant fraction, cardiopulmonary exercise testing to quantify functional reserve, and joint co-evaluation by ACHD and PH specialists, in line with the 2022 ESC/ERS PH guidelines and AHA/ACC ACHD guidelines. The o*ptimization phase*, ideally 4 to 12 weeks before surgery, should focus on initiation or escalation of PAH-targeted therapy when pulmonary vascular resistance is elevated, pulmonary rehabilitation, smoking cessation, and treatment of obstructive sleep apnea when present. The *intraoperative management* requires transesophageal echocardiography, pulmonary artery pressure monitoring, immediate availability of inhaled nitric oxide and prostacyclin analogues, and a low threshold for institution of mechanical circulatory support including veno-arterial extracorporeal membrane oxygenation in severe PH. The *postoperative phase* should involve intensive care unit monitoring with continuous hemodynamic assessment, early extubation when feasible, uninterrupted PH-directed therapy, and daily multidisciplinary rounds with ACHD, PH, cardiac intensive care, and surgical teams. Finally, *discharge and follow-up* at 2, 6, and 12 weeks in a combined ACHD-PH clinic should cover medication reconciliation, cardiac rehabilitation referral, and re-evaluation of PH therapy. Such pathway-based care, delivered at high-volume centers, is what is most likely to translate the LOS reductions and resource-utilization gains suggested by our findings into measurable clinical benefits.

Future research priorities include prospective evaluation of PH-targeted therapy optimization before PVR, identification of hemodynamic and imaging thresholds that define a reversible versus non-reversible RV phenotype, and development of ACHD- and PH-specific perioperative risk scores that outperform generic surgical scores. Longitudinal studies capturing late mortality, rehospitalization, reintervention, and quality of life will be essential to determine whether the short-term parity in mortality between PH and non-PH patients persists over time.

Limitations

Our study's strength lies in its national scope and real-world applicability, capturing the true spectrum of PVR patients across diverse healthcare settings. However, the retrospective design and reliance on administrative coding introduce inherent limitations. The study period (2018–2020) spans the onset of the COVID-19 pandemic, and we did not examine year-to-year differences or hospital-level factors (volume, academic status, urban vs rural) that might influence outcomes. Definitions and grading of PH were not fully standardized, and detailed hemodynamic data, PH-directed treatment regimens, use of mechanical circulatory support, and ICU management strategies were not uniformly available. Outcomes were restricted to in-hospital events, precluding assessment of 30-day or long-term mortality, functional status, and patient-reported outcomes. Finally, although multivariable models adjusted for major measured confounders, residual confounding by unmeasured factors cannot be excluded. Future prospective or registry-based studies with granular clinical data will be essential to definitively characterize mortality risks and validate risk stratification models.

A few caveats are worth noting. The ICD-10-CM codes we used to identify PH (I27.0 and I27.2x) do not differentiate between WHO clinical groups of PH, do not capture hemodynamic severity, and provide no information on PH-directed therapy at the time of surgery. So, the severity of PH and how well it was being treated are confounders we cannot account for. The procedure codes have a related issue. The ICD-10-PCS codes used (02RH0KZ, 02RH3KZ, and 02RH4KZ) are applied to both surgical and transcatheter PVR, and we cannot consistently separate the two from administrative data alone. This matters because Melody and Sapien XT/S3 valves were already in routine use during 2018-2020, even though Harmony and Alterra had not yet been FDA-approved. At most ACHD centers in that period, surgical PVR was being reserved for patients who were not good candidates for transcatheter PVR, such as those with native or large outflow tracts, complex anatomy, or a need for concomitant surgery, so our surgical cohort is likely enriched for higher-complexity cases. Finally, although TOF was the most common diagnosis (32%), we could not fully characterize the underlying lesion mix, the indication for PVR, or concomitant procedures, all of which influence surgical risk. Stratification by surgical versus

transcatheter approach and by underlying anatomy will be an important focus of future registry-based work.

Conclusions

Pulmonary hypertension in the PVR setting defines a small but distinctly high-risk phenotype, older, predominantly female, and burdened by significant pulmonary comorbidity, in whom hospitalization is approximately 50% longer after adjustment with important implications for resource utilization, despite no clear increase in in-hospital mortality. This dissociation between survival and resource use positions PH not as a contraindication to PVR, but as a powerful marker of clinical complexity that should prompt earlier intervention before the mid-40s, when RV and pulmonary vascular remodeling may still be reversible. The disproportionate effect of emphysema on length of stay further underscores the central role of lung disease, reinforcing the need for meticulous preoperative assessment, aggressive optimization of pulmonary status, and structured perioperative pathways delivered within specialized ACHD–PH programs capable of intensive, protocolized management.

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Table 1: Table showing the demographic and clinical characteristics comparing patients undergoing pulmonary valve replacement, with and without pulmonary hypertension (PH).

Characteristics	Overall N=1533[1]	No PH N=1395[1]	PH N=138[1]	p-value[2]
Age (in years)	27±19	26±18	39±22	<0.001
Length of stay (in days)	21	14	7	<0.001
Sex				0.004
Male	904 (59%)	839 (60%)	65 (47%)	
Female	629 (41%)	556 (40%)	73 (53%)	
In-hospital death				0.004
Yes	20 (1.3%)	14 (1.0%)	6 (4.3%)	
No	1513 (98.7%)	1381 (99%)	132 (96.7%)	
Obesity				0.10
Yes	165 (11%)	144 (10%)	21 (15%)	
No	1368 (89%)	1251 (90%)	117 (85%)	
Hypertension				>0.9
Yes	219 (14%)	200 (14%)	19 (14%)	
No	1314 (86%)	1195 (86%)	119 (86%)	
Diabetes Mellitus				0.059
Yes	85 (5.5%)	72 (5.2%)	13 (9.4%)	
No	1448 (94.5%)	1323 (94.8%)	125 (90.6%)	
Asthma				0.4
Yes	120 (7.8%)	106 (7.6%)	14 (10%)	
No	1413 (92.2%)	1289 (92.4%)	124 (90%)	

Chronic Kidney Disease				0.2
Yes	116 (7.5%)	104 (7.4%)	12 (8.7%)	
No	1417 (92.5%)	1291 (92.6%)	116 (91.3%)	
Chronic Obstructive Pulmonary Disease				<0.001
Yes	30 (2%)	18 (1.3%)	12 (8.7%)	
No	1503 (98%)	1377 (98.7%)	126 (91.3%)	
Emphysema				0.005
Yes	6 (0.4%)	3 (0.2%)	3 (2.2%)	
No	1527 (99.6%)	1392 (98.8%)	135 (97.8%)	

[1]Mean ± SD; Median (Min, Max); n(%)

[2]Welch Two Sample t-test; Wilcoxon rank sum test; Pearson's Chi-square test

PH: Pulmonary hypertension

Figure Legends:

Figure 1: Adjusted predictors of in-hospital mortality. Forest plot of odds ratios (log scale) with 95% confidence intervals from the Firth logistic regression model for age, sex, pulmonary hypertension, and comorbidities.

Figure 2: Receiver operating characteristic (ROC) curve for age predicting in-hospital mortality. ROC curve showing the discriminative ability of age after pulmonary valve replacement, with an optimal cutoff of 45.5 years (sensitivity 0.65, specificity 0.82).

Figure 3a: Length of stay (LOS) by pulmonary hypertension (PH) status. Median postoperative LOS in days for patients with and without PH undergoing pulmonary valve replacement, shown as color-matched box-and-whisker plots with individual data points.

Figure 3b: Effect of pulmonary hypertension (PH) and comorbidities on hospital length of stay (LOS). Forest plot of adjusted percent change in LOS from log-linear regression, with 95% confidence intervals for PH, age, sex, and major comorbidities.

Figures

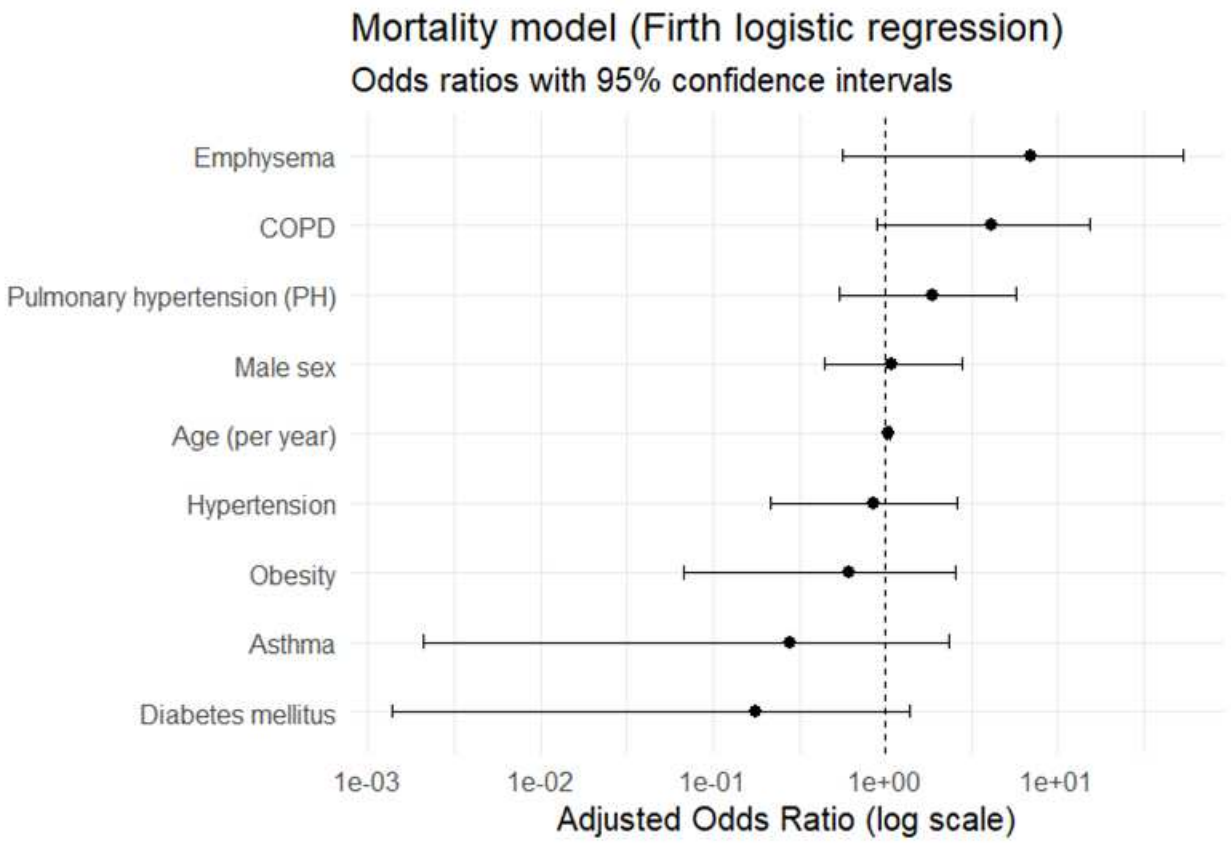


Figure 1.tiff

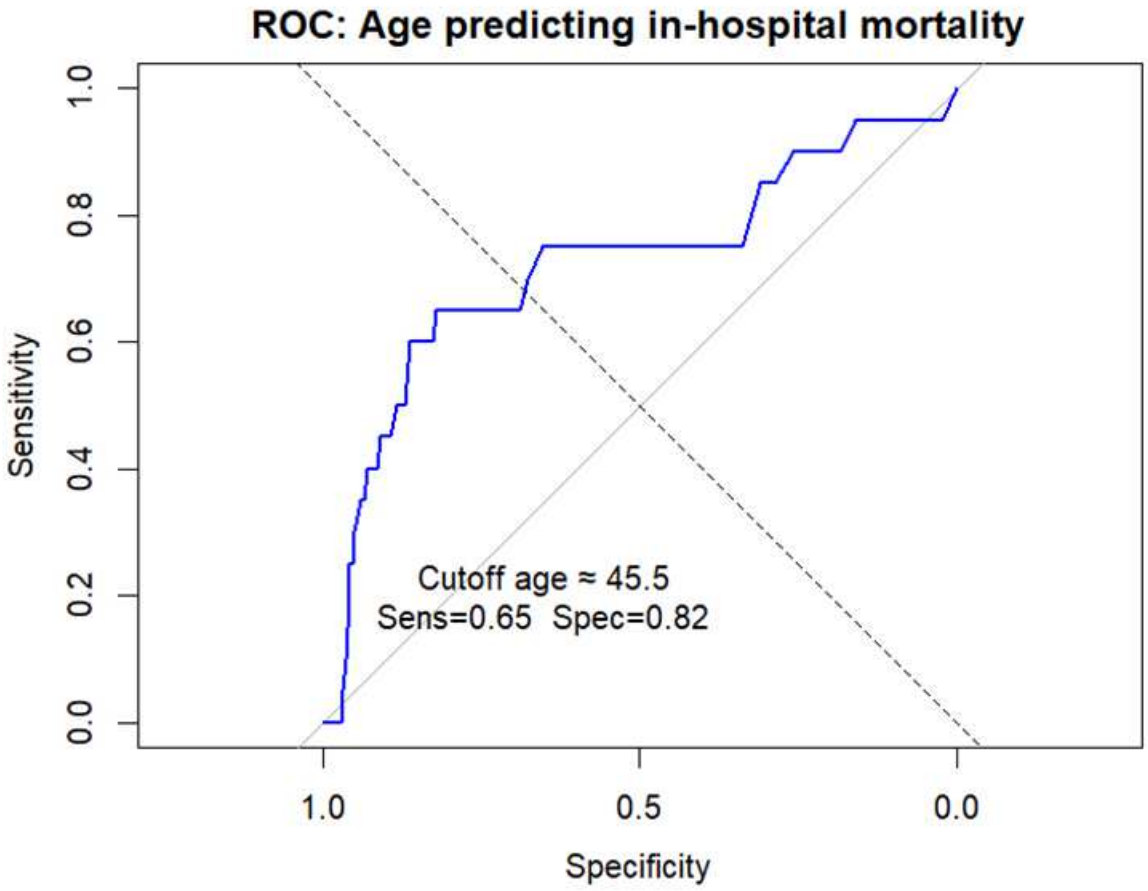


Figure 2.tiff

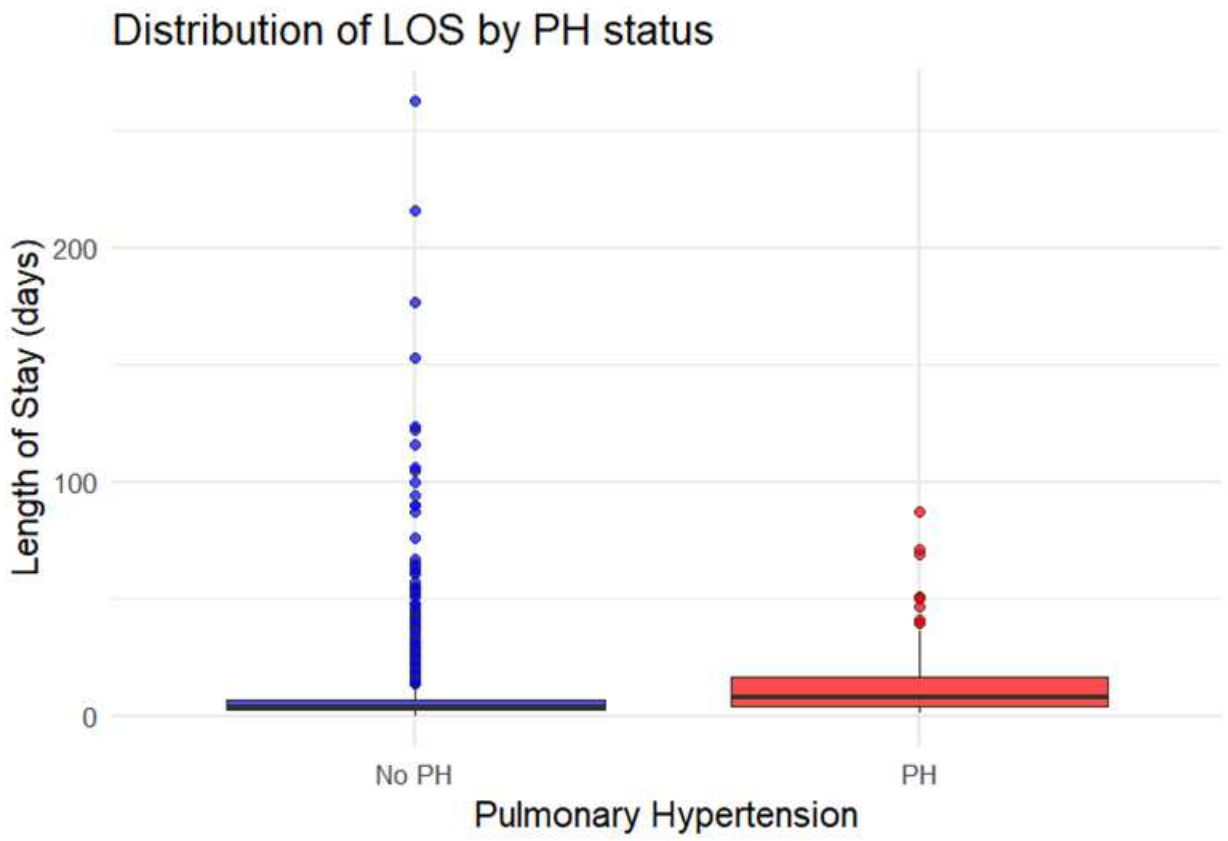


Figure 3a.tiff

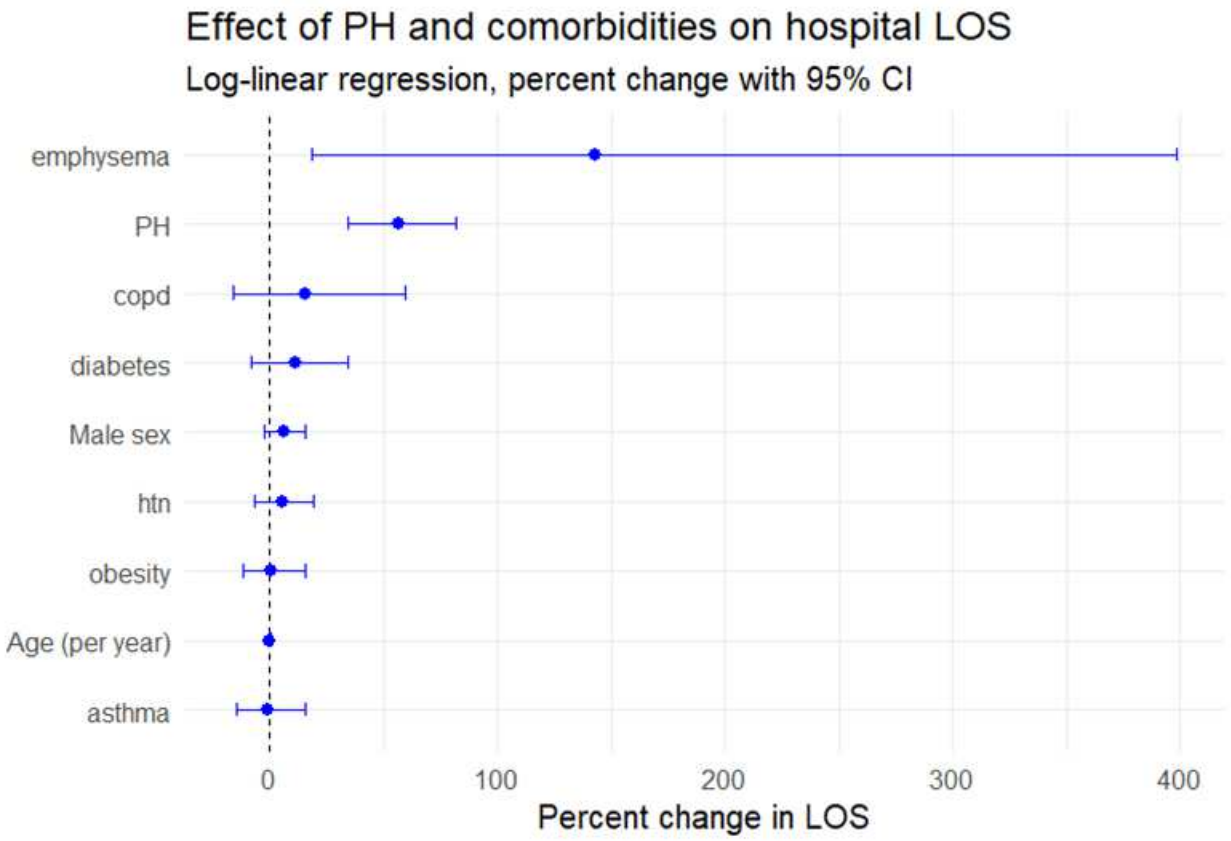


Figure 3b.tiff

Supplemental Table S1

Table. ICD-10 codes used for analysis

Condition / Procedure	ICD-10 Code(s)	Description
Pulmonary Hypertension	I27.0	Primary pulmonary hypertension
	I27.2	Other secondary pulmonary hypertension
	I27.20	Pulmonary hypertension, unspecified
	I27.21	Secondary pulmonary arterial hypertension
Pulmonary Valve Replacement	02RH0KZ	Replacement of pulmonary valve with synthetic substitute, open approach
	02RH3KZ	Replacement of pulmonary valve with synthetic substitute, percutaneous approach
	02RH4KZ	Replacement of pulmonary valve with synthetic substitute, percutaneous endoscopic approach
Hypertension	I10	Essential (primary) hypertension
Diabetes Mellitus	E11.xx	Type 2 diabetes mellitus (all subcategories)
Chronic Obstructive Pulmonary Disease (COPD)	J44.x	Chronic obstructive pulmonary disease (all subcategories)
Emphysema	J43.9	Emphysema, unspecified
Obesity	E66.x	Overweight and obesity (all subcategories)
Asthma	J45.x	Asthma (all subcategories)

".xx" indicates inclusion of all subcodes under that ICD-10 category (e.g., E11.0–E11.9 for type 2 diabetes mellitus; J45.20–J45.99 for asthma).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementalTable.docx