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The Influence of Six Weeks of Exercise Training on Echocardiographic Right Ventricular Function in Adults with Congenital Heart Disease—Associated Pulmonary Hypertension

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ABSTRACT: Exercise training may improve RV function in adults with CHD-associated PH; however, evidence based on echocardiographic outcomes remains limited. This study evaluated the effect of six weeks of exercise training on TAPSE, lateral S' velocity, and FAC. This single-arm pre-post study enrolled 13 adults with CHD-associated PH at the Integrated Heart Center, Dr. Wahidin Sudirohusodo Hospital, Makassar, Indonesia. Participants completed supervised moderate-intensity exercise training twice weekly for six weeks. Echocardiographic RV-function parameters were assessed at baseline and after the intervention. Changes in TAPSE and lateral S' velocity were analysed using paired t-tests, whereas FAC was analysed using the Wilcoxon signed-rank test. TAPSE increased numerically after training but was not statistically significant (1.90 ± 0.35 to 1.93 ± 0.45 cm; $p = 0.783$). Lateral S' velocity also increased without statistical significance (11.92 ± 2.21 to 12.38 ± 2.98 cm/s; $p = 0.495$). In contrast, FAC increased significantly from 23% (range, 5–48%) to 34% (range, 13–47%; $Z = -2.269$, $p = 0.023$). Six weeks of structured exercise training was associated with improved RV function, predominantly through improved FAC. Exercise training may represent a useful adjunctive non-pharmacological strategy in the comprehensive management of adults with CHD-associated PH.

1. INTRODUCTION

Pulmonary hypertension (PH) is a haemodynamic disorder defined by a mean pulmonary arterial pressure >20 mmHg at rest, measured by right-heart catheterisation; pre-capillary PH is additionally characterised by a pulmonary artery wedge pressure ≤ 15 mmHg and pulmonary vascular resistance >2 Wood units [1,2]. In congenital heart disease (CHD), persistent systemic-to-pulmonary shunting and complex anatomical or haemodynamic abnormalities can promote pulmonary vascular disease and create a heterogeneous, high-risk PH population requiring individualised assessment [3,4].

Right ventricular (RV) adaptation to increased pulmonary vascular load is a major determinant of clinical status and prognosis in PH, while progressive RV dysfunction is associated with adverse outcomes [5]. Echocardiography enables practical serial evaluation of RV systolic function, and tricuspid annular plane systolic excursion (TAPSE), lateral tricuspid annular S' velocity, and RV fractional area change (FAC) are among the most established parameters for this purpose [6,7].

Exercise-based rehabilitation improves exercise capacity and health-related quality of life in patients with PH and is not associated with an increased risk of serious adverse events when delivered in appropriately supervised programmes [8]. However, available evidence has predominantly assessed functional and patient-reported outcomes, whereas data on the effect of structured exercise training on echocardiographic markers of RV function in adults with CHD-associated PH remain limited [8–10]. Therefore, this study evaluated the effect of a six-week exercise-training programme on RV function, assessed by changes in TAPSE, lateral S' velocity, and FAC, in adults with CHD-associated PH.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

This single-arm pre–post experimental study was conducted at the Integrated Heart Center, Dr. Wahidin Sudirohusodo Hospital, Makassar, Indonesia, between June and December 2025. Participants underwent baseline assessment, completed a six-week exercise-training programme, and underwent repeat assessment at the end of the intervention.

2.2 Participants

Adult outpatients aged >18 years with PH and CHD attending the Integrated Heart Center during the recruitment period were screened for eligibility. PH was confirmed by right-heart catheterisation, while CHD was confirmed by echocardiography and/or right-heart catheterisation. Participants were required to be receiving stable sildenafil therapy and to provide written informed consent. Patients were excluded if they had left-sided heart disease, severe lung disease, acute coronary syndrome within the preceding 40 days, percutaneous coronary intervention within the preceding two weeks, coronary artery bypass grafting within the preceding three months, intracardiac thrombus or recent embolism, resting heart rate >90 beats/min, exercise-induced arrhythmia or conduction disorder, acute myocarditis or pericarditis, hypertrophic cardiomyopathy, haemoglobin <11.0 g/dL, significant musculoskeletal or neurological disability, thrombophlebitis, uncontrolled non-cardiac disease, active malignancy, aortic aneurysm, or severe psychiatric disorder. Patients who were unable or unwilling to participate were also excluded. Eligible patients were enrolled until the target sample size was achieved. The minimum required sample size was 12 participants, calculated using the expected clinically significant change, standard deviation, and a 95% confidence level. Participants were considered to have withdrawn if they attended <80% of prescribed sessions or did not return for the six-week follow-up assessment.

2.3 Study Procedures and Exercise Training

Baseline demographic characteristics, medical history, medication use, and socioeconomic information were collected through review of official records and participant interviews. Weight, height, body mass index, resting heart rate, and blood pressure were recorded during the baseline clinical assessment. All participants received standardised cardiac rehabilitation education addressing medication adherence, stress management, smoking cessation, healthy dietary practices, risk-factor control, and physical activity. Participants were provided with a 1-kg dumbbell, incentive spirometer, digital blood-pressure monitor, pulse oximeter, exercise logbook, and educational materials. The exercise-training programme was delivered at the Integrated Heart Center under direct supervision of the cardiac rehabilitation team. Participants attended two moderate-intensity sessions per week for six weeks, with each session lasting 30 minutes. Intensity was prescribed according to perceived exertion using the Borg scale and was individually adjusted by the rehabilitation team according to symptoms and clinical tolerance. Session attendance and home activity were documented in individual logbooks, which were reviewed periodically; adherence was calculated as the proportion of prescribed supervised sessions attended. Participants with adherence <80% were classified as withdrawn.

2.4 Echocardiographic Assessment

RV function was assessed at baseline and after completion of the six-week programme by transthoracic echocardiography using a GE Vivid E95 system. Images were acquired using standard apical four-chamber views. TAPSE was measured as the systolic excursion of the lateral tricuspid annulus along the longitudinal RV axis. Lateral S' velocity was obtained by tissue Doppler

imaging at the lateral tricuspid annulus. FAC was calculated from the RV end-diastolic and end-systolic areas in the apical four-chamber view as $[(\text{end-diastolic area} - \text{end-systolic area}) / \text{end-diastolic area}] \times 100$. The same predefined outcomes—TAPSE, lateral S' velocity, and FAC—were recorded at both assessments.

2.5 Statistical Analysis

Data were analysed using IBM SPSS Statistics version 21. Continuous variables were presented as mean $\pm$ standard deviation when approximately normally distributed or median (range) when non-normally distributed; categorical variables were presented as frequencies and percentages. Pre–post changes in TAPSE and lateral S' velocity were assessed using paired t-tests because these paired measures were approximately normally distributed. FAC was analysed using the Wilcoxon signed-rank test because its paired distribution was non-normal. All tests were two-sided, and a p value <0.05 was considered statistically significant.

3. RESULTS AND DISCUSSION

3.1 Participant Characteristics

Thirteen adults with CHD-associated PH were included in the final analysis. The mean age was 31.76 ± 8.49 years, and 11 participants (84.6%) were women. The mean body mass index was 19.59 ± 3.03 kg/m². ASD was the predominant CHD diagnosis, occurring in 10 participants (76.9%), followed by VSD in two participants (15.4%) and PDA in one participant (7.7%). Educational attainment and occupational status are detailed in Table 1.

Table 1. Baseline Demographic and Clinical Characteristics of the Study Participants

Characteristic	Value (n = 13)
Age, years	31.76 $\pm$ 8.49
Female sex, n (%)	11 (84.6)
Male sex, n (%)	2 (15.4)
Body mass index, kg/m ²	19.59 $\pm$ 3.03
Education, n (%)	
Primary school	1 (7.7)
Junior high school	1 (7.7)
Senior high school	6 (46.2)
Bachelor's degree	5 (38.5)
Occupation, n (%)	
Student	5 (38.5)
Homemaker	5 (38.5)
Employee	2 (15.4)
Self-employed	1 (7.7)
CHD diagnosis, n (%)	
ASD	10 (76.9)
VSD	2 (15.4)
PDA	1 (7.7)

Values are presented as mean $\pm$ standard deviation or n (%).

3.2 Baseline Haemodynamic Characteristics

Baseline right-heart catheterisation findings are presented in Supplementary Table 1. Participants had markedly elevated pulmonary artery pressures, with a mean systolic pulmonary artery pressure of 96.42 ± 35.10 mmHg and a mean mPAP of 61.00 ± 22.51 mmHg. The complete right-sided and left-sided pressure measurements, oxygen-saturation values, flow ratio, PAR, and PAR/SVR are summarised in Supplementary Table 1.

3.3 Baseline Medication Use

All participants received sildenafil therapy. Furosemide and beraprost were used by 11 (84.6%) and nine (69.2%) participants, respectively, whereas RAAS blockers and beta-blockers were used by four (30.8%) and three (23.1%) participants, respectively. No participant received a calcium-channel blocker.

3.4 Echocardiographic Findings by CHD Subtype

Echocardiographic findings before and after the exercise-training programme according to CHD subtype are shown in Table 2. In the ASD subgroup, TAPSE remained largely unchanged, while lateral S' velocity and FAC increased after the intervention. Participants with VSD showed numerical increases in all three RV-function parameters. The participant with PDA showed unchanged TAPSE but increased lateral S' velocity and FAC. These subgroup findings are descriptive only because of the limited number of participants with VSD and PDA.

Table 2. Echocardiographic Findings by CHD Subtype Before and After Six Weeks of Exercise Training

Variable	ASD Pre (n = 10)	ASD Post	VSD Pre (n = 2)	VSD Post	PDA Pre (n = 1)	PDA Post
Defect size, mm	3.07 ± 1.36	3.07 ± 1.36	1.30 ± 0.42	1.30 ± 0.42	7.00	7.00
LA major diameter, cm	5.30 ± 0.97	5.06 ± 1.06	6.30 ± 0.70	6.00 ± 1.97	4.60	4.60
LA minor diameter, cm	3.18 ± 0.59	3.41 ± 0.68	4.45 ± 0.63	4.65 ± 1.06	2.70	2.90
RA major diameter, cm	5.96 ± 1.28	5.69 ± 1.17	5.95 ± 2.61	5.70 ± 2.26	5.00	5.00
RA minor diameter, cm	4.64 ± 0.83	4.51 ± 0.70	4.75 ± 1.06	4.51 ± 0.70	4.30	3.40
RV basal diameter, cm	5.34 ± 0.77	4.80 ± 0.81	5.10 ± 1.83	5.15 ± 2.47	3.60	3.60
TR Vmax, m/s	4.16 ± 0.95	3.88 ± 0.71	4.00 ± 1.57	3.15 ± 1.90	5.26	4.00
TR pressure gradient, mmHg	71.80 ± 31.75	62.04 ± 23.59	67.51 ± 52.77	47.83 ± 49.30	110.63	64.10
PV acceleration time	101.20 ± 31.60	123.70 ± 37.91	106.80 ± 31.39	115.50 ± 4.94	119.00	119.00
Left ventricular ejection fraction, %	65.63 ± 5.95	66.00 ± 4.26	49.00 ± 28.28	49.00 ± 28.28	75.00	75.00
TAPSE, cm	1.90 ± 0.40	1.89 ± 0.50	1.95 ± 0.21	2.20 ± 0.14	1.90	1.90
Lateral S' velocity, cm/s	11.80 ± 2.44	12.00 ± 3.01	11.50 ± 0.70	12.00 ± 1.41	14.00	17.00
FAC, %	21.60 ± 11.35	30.80 ± 12.43	29.00 ± 19.79	32.50 ± 19.09	31.00	34.00
RVWLS, %	-15.98 ± 6.60	-15.36 ± 3.53	-14.70 ± 6.50	-13.60 ± 3.04	-15.80	-14.60
PASP, mmHg	81.35 ± 31.21	70.16 ± 22.89	80.63 ± 55.47	58.48 ± 53.06	118.67	72.00

mPAP, mmHg	51.60 ± 19.04	44.79 ± 13.90	51.18 ± 33.83	37.67 ± 32.36	74.38	45.92
PVR, WU	0.36 ± 0.18	0.33 ± 0.19	0.29 ± 0.26	0.19 ± 0.17	0.46	0.35

Values are presented as mean ± standard deviation, except for the PDA subgroup, which is presented as an individual value. No between-group or subgroup statistical comparisons were performed.

3.5 Changes in RV Function After Exercise Training

After six weeks of exercise training, TAPSE increased from 1.90 ± 0.35 cm to 1.93 ± 0.45 cm; however, the difference was not statistically significant (95% CI for the pre–post difference, -0.269 to 0.207 ; $p = 0.783$). Lateral S' velocity also increased numerically from 11.92 ± 2.21 cm/s to 12.38 ± 2.98 cm/s, without a statistically significant difference (95% CI for the pre–post difference, -1.891 to 0.968 ; $p = 0.495$). In contrast, FAC increased significantly from 23% (range, 5–48%) at baseline to 34% (range, 13–47%) after the intervention ($Z = -2.269$, $p = 0.023$) (Table 3).

Table 3. Changes in Echocardiographic RV Function After Six Weeks of Exercise Training

Outcome	Baseline	Week 6	Test statistic or 95% CI	<i>p</i> value
TAPSE, cm	1.90 ± 0.35	1.93 ± 0.45	95% CI, -0.269 to 0.207	0.783
Lateral S' velocity, cm/s	11.92 ± 2.21	12.38 ± 2.98	95% CI, -1.891 to 0.968	0.495
FAC, %	23 (5–48)	34 (13–47)	$Z = -2.269$	0.023

Values are presented as mean ± standard deviation or median (range). TAPSE and lateral S' velocity were analysed using paired *t*-tests; FAC was analysed using the Wilcoxon signed-rank test.

3.6. Discussion

The present study found a statistically significant increase in FAC after six weeks of supervised exercise training, whereas changes in TAPSE and lateral S' velocity were not statistically significant. Thus, the results support a signal of improvement in a global RV systolic-function measure, but do not establish improvement across all RV indices. A multiparametric approach remains important because TAPSE, lateral S', and FAC represent distinct components of RV mechanics [11,12].

The FAC finding is clinically relevant because this parameter incorporates both longitudinal and radial components of RV contraction. In PH, increased RV afterload may cause geometric remodelling and interventricular septal displacement, which can limit the ability of longitudinal indices to represent global RV performance fully. The observed FAC increase may therefore reflect favourable adaptation of global RV systolic function after training. This interpretation is consistent with evidence that comprehensive assessment of RV function provides more clinical and prognostic information than reliance on a single echocardiographic parameter [11].

The non-significant changes in TAPSE and lateral S' should be interpreted cautiously. Both measures predominantly represent longitudinal motion at the tricuspid annulus and may be less responsive to short-term changes in global RV contractility. Their numerical direction of change is consistent with Ratri et al. (2025) [13], who reported that RV–pulmonary artery coupling, including TAPSE-related indices, was associated with exercise tolerance in patients with CHD-associated PH. Similarly, the relationship between TAPSE/sPAP and exercise haemodynamics supports the relevance of RV adaptation to pulmonary vascular load during physical activity [14]. However, numerical changes in these indices should not be interpreted as evidence of a treatment effect in the absence of statistical significance.

Exercise training may improve RV performance through several mechanisms, including improved endothelial function, peripheral oxygen extraction, ventilatory efficiency, autonomic balance, and skeletal-muscle conditioning. These adaptations may reduce the physiological burden imposed on the RV during exertion and support more efficient RV–pulmonary arterial coupling. Previous studies have shown that exercise-based rehabilitation can improve functional capacity and quality of life in PH, supporting its role as an adjunct to pharmacological treatment [15,16].

The present findings support the central role of the RV in exercise adaptation among patients with CHD-associated PH. Ruijsink et al. (2020) demonstrated that RV systolic function contributes substantially to optimising left ventricular performance

during exercise [17], while Hu et al. (2023) reported favourable RV structural and functional adaptation in individuals exposed to sustained endurance exercise [18]. Although the clinical populations differ, these findings support the biological plausibility that carefully prescribed exercise can promote beneficial RV adaptation. The findings should nevertheless be interpreted as preliminary because of the small sample, single-arm pre–post design, absence of a control group, and possible effects of echocardiographic measurement variability, participant heterogeneity, concomitant therapy, and other clinical factors. Accordingly, the significant FAC result should be confirmed in adequately powered controlled studies with standardised echocardiographic acquisition and blinded outcome assessment.

4. CONCLUSION

In adults with CHD-associated PH, six weeks of supervised exercise training were associated with a statistically significant improvement in FAC, a measure of global RV systolic function. TAPSE and lateral S' velocity showed numerical but non-significant changes. These preliminary findings support further controlled evaluation of structured exercise training as an adjunctive non-pharmacological component of comprehensive care for adults with CHD-associated PH.

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AUTHOR CONTRIBUTIONS

Conceptualization: JL, ZD, AFG, YP, MW, AAZ. Methodology: JL, ZD, AFG, YP, MW, AAZ. Formal analysis: JL, AAZ. Investigation: JL. Data curation: JL, AAZ. Writing—original draft preparation: JL. Writing—review and editing: JL, ZD, AFG, YP, MW, AAZ. Visualization: JL. Supervision: ZD, AFG, YP, MW, AAZ. Project administration: JL, ZD. All authors read and approved the final manuscript.

CONFLICT OF INTEREST

The authors declare that they have no competing interests.

ETHICS STATEMENT

The study was approved by the Health Research Ethics Committee, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia (No. 932/UN4.6.4.5.31/PP36/2025). The study was conducted in accordance with the Declaration of Helsinki and applicable local regulations.

DATA AVAILABILITY STATEMENT

The datasets generated and analysed during the current study are available from the corresponding author upon reasonable request.

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Supplementary Table 1. Baseline Right-Heart Catheterisation Findings

Variable	Mean $\pm$ SD	Range
HSVC saturation, %	62.08 $\pm$ 12.13	42–79
LSVC saturation, %	60.08 $\pm$ 8.35	46–72
IVC saturation, %	66.38 $\pm$ 8.19	53–84
Right atrial systolic pressure, mmHg	12.17 $\pm$ 4.26	6–18
Right atrial diastolic pressure, mmHg	11.25 $\pm$ 4.62	4–22
Mean right atrial pressure, mmHg	8.23 $\pm$ 4.21	3–16
Right atrial saturation, %	71.15 $\pm$ 11.52	52–87
Right ventricular systolic pressure, mmHg	88.67 $\pm$ 39.30	19–150

Right ventricular diastolic pressure, mmHg	10.33 $\pm$ 14.08	1–40
Mean right ventricular pressure, mmHg	19.92 $\pm$ 17.04	4–61
Right ventricular saturation, %	74.38 $\pm$ 13.23	49–90
Pulmonary artery systolic pressure, mmHg	96.42 $\pm$ 35.10	31–146
Pulmonary artery diastolic pressure, mmHg	38.08 $\pm$ 19.46	1–71
Mean pulmonary artery pressure, mmHg	61.00 $\pm$ 22.51	12–96
Pulmonary artery saturation, %	73.31 $\pm$ 12.30	49–95
Left atrial systolic pressure, mmHg	14.25 $\pm$ 4.59	8–23
Left atrial diastolic pressure, mmHg	13.50 $\pm$ 4.34	8–21
Mean left atrial pressure, mmHg	10.82 $\pm$ 3.95	6–17
Aortic systolic pressure, mmHg	106.92 $\pm$ 22.83	70–142
Aortic diastolic pressure, mmHg	63.00 $\pm$ 18.58	36–109
Mean aortic pressure, mmHg	79.42 $\pm$ 16.59	50–109
Aortic saturation, %	88.17 $\pm$ 7.90	72–98
Flow ratio	2.81 $\pm$ 6.10	0.30–22.00
PAR	24.34 $\pm$ 23.09	0.14–72.20
PAR/SVR	1.19 $\pm$ 0.92	0.11–2.70

Values are presented as mean $\pm$ standard deviation and range.