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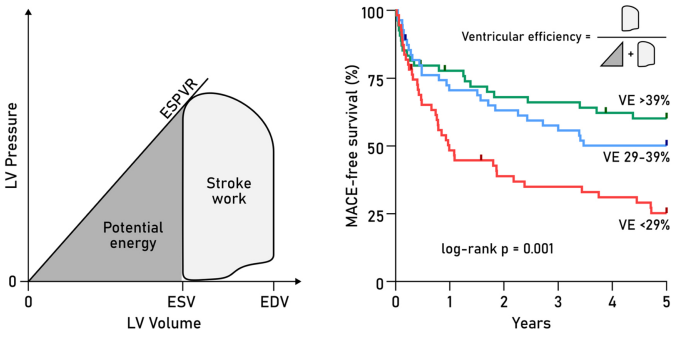
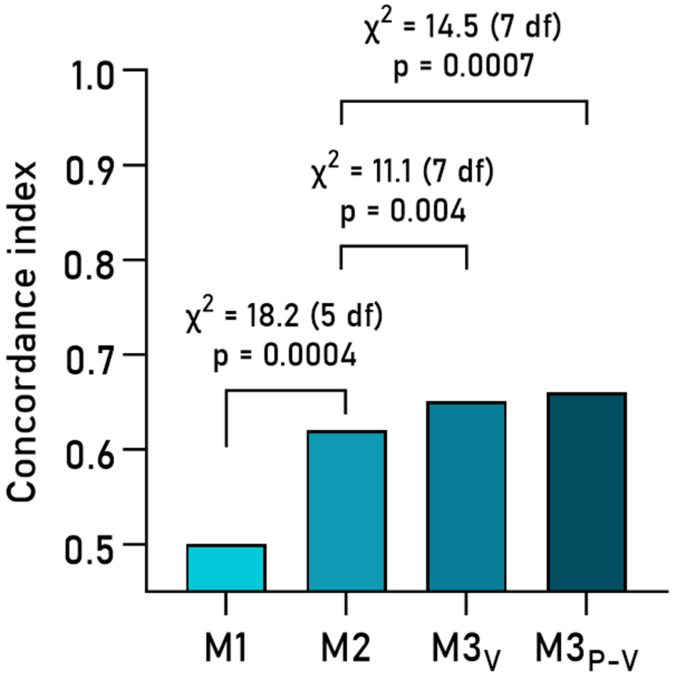
endpoint was time to MACE within 5 years. Associations with clinical outcomes were evaluated using multivariate Cox regressions.

Results: 88 patients (54%) experienced at least one MACE (CV death n = 23, HF hospitalization n = 49, revascularization n = 8, myocardial infarction n = 6, cardiac arrest n = 1, ventricular tachycardia n = 1). Univariate Cox regressions found significant associations with the primary endpoint for the PV loop parameters stroke work, ventricular efficiency, external power, contractility, and energy per ejected volume, alongside HF etiology, EF, global longitudinal strain, and NT-proBNP level. In iterative multivariate Cox regression adjusted for age, sex, hypertension, diabetes, and HF etiology (figure 2), ventricular efficiency was found to predict MACE, with hazard ratio 1.04 (95% CI: 1.01-1.08) per-% decrease, p = 0.01.

Conclusion: Ventricular efficiency, derived from non-invasive pressure-volume loop analysis from standard CMR scans, independently predicts major adverse cardiac events in patients with HFrEF.





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Kiosk 10R-FB-07

Pre- and Post-treatment CMR Measurement Changes in Pulmonary Arterial Hypertension

Samer Alabed, MD, PhD[1], Pankaj Garg, MD, PhD[2], Krit Dwivedi, MD, PhD[1], Ahmed Maiter, MD[1], Mahan salehi, MD[1], Rebecca gosling, MD, PhD[1], Michael Sharkey, BEng[1], Rob Van Der Geest, PhD[3], David Kiely, MD, PhD[1], Andrew Swift, MD, PhD[1]

[1] *University of Sheffield*
[2] *University of East Anglia*
[3] *Leiden University Medical Center*

Background: Pulmonary Arterial Hypertension (PAH) has a poor prognosis and survival is determined primarily by right ventricular function. CMR is the gold standard technique for assessing biventricular volumes and function. CMR is increasingly considered an endpoint in clinical studies and this study aims to quantify the change in CMR measurements pre- and post-treatment in PAH.

Methods: Consecutive treatment-naïve patients with pulmonary arterial hypertension between 2010 and 2022 who had two CMR scans (at baseline prior to treatment and 12 months following treatment) were identified from the ASPIRE registry. CMR was performed with 1.5 Tesla MRI systems from GE. CMR measurements were obtained through an automated and validated segmentation tool. A dependent t-test between pre-and post-treatment was performed with a significance threshold of 0.05.

Results: 254 patients with PAH were included (aged 53 ± 16 years, 79% female, and 66% categorised as intermediate risk based on 2022 ESC/ERS risk score. Between the two scans, patients were treated with phosphodiesterase 5 inhibitors (86%), endothelin receptor antagonists (72%), parenteral prostanoid (17%) and other medications (4%), with 32% receiving monotherapy, 48% dual combination and 20% triple combination therapy. All CMR measurements showed statistically significant differences pre- and post-treatment. The largest differences were a reduction of right ventricular end-systolic volume (RVESV) by 20 ml and an increase of left ventricular end-diastolic volume (LVEDV) by 22 ml. The change in right ventricular ejection fraction (RVEF) was 7%. Changes in CMR measurements are shown in Table 1.

Conclusion: CMR is sensitive to changes post-treatment in PAH. This study provides further support for the use of CMR as a PAH clinical trial endpoint and will aid trial size calculations for studies using CMR.

Pre - and post- treatment cardiac MRI measurements.

	Pre -Treatment	*Post-Treatment*	*Absolutedifference*
RVEF (%)	34 ± 12	41 ± 11	7 ± 10
RVESV (ml)	139 ± 61	119 ± 59	-20 ± 39
RVEDV (ml)	205 ± 62	195 ± 68	-9 ± 44
RVSV (ml)	66 ± 24	76 ± 28	10 ± 23
RVEDM (g)	51 ± 16	48 ± 15	-3 ± 10
LVEF (%)	54 ± 9	58 ± 8	3 ± 9
LVESV (ml)	52 ± 19	58 ± 20	6 ± 14
LVEDV (ml)	114 ± 30	135 ± 35	22 ± 25
LVSV (ml)	61 ± 19	77 ± 21	16 ± 19
LVEDM (g)	86 ± 21	89 ± 22	3 ± 10

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Kiosk 10R-FB-08
Pulmonary Blood Volume in Healthy Volunteers and Heart Failure Patients at Rest and During Exercise

Eric Morgan, MD, PhD[1], Charles Benton, RT[1], Shahrad Shadman, MD[1], Gaby Weissman, MD[2], Ana Barac, MD[3], Marcus Carlsson, MD, PhD[1]

[1] *National Heart, Lung, and Blood Institute, National Institutes of Health*
[2] *Medstar Washington Medical Center*
[3] *INOVA*

Background: Increased pulmonary blood volume (PBV), the intravascular volume in the pulmonary circuit extending from the origin of the pulmonary artery to the orifice of the pulmonary veins, has been shown to have prognostic value in heart failure (HF) patients. It is not known if PBV changes with exercise in HF patients with reduced cardiac function. Thus, we sought to quantify PBV in HF patients at rest and during exercise and compare these results with those obtained in healthy volunteers (HV).

Methods: PBV was quantified in 10 HV and 7 HF Patients with EF< 55% using MRI as previously described. Briefly, pulmonary transit time (PTT) was quantified via first pass perfusion in the short axis view and taken as the time between the center of gravity of the time intensity curves of the right and left ventricles. The PTT was then multiplied by the flow through the pulmonary artery. Pulmonary arterial flows and PTT were obtained in each subject both at rest and during exercise. Real time phase contrast imaging of the pulmonary artery was used during the exercise portion while standard 2D phase contrast imaging was used during the rest period. Exercise was achieved by peddling on a stationary ergometer, and exercise intensity was gradually increased until the target heart rate (220-age (years) X 70%) was reached or until patients reported the onset of fatigue. Data were analyzed using two-tailed paired t-tests and are presented as the mean ± standard error of the mean.

Results: Patients were 57% female with a mean age of 55 years. HV were 40% female with a mean age of 43 years. Mean LVEF in HV was 61% (range 56%-70%) and in Patients was 39% (range 30%-53%). There was no difference in heart rate in the HV or Patients at rest (60 ± 4 vs. 66 ± 5 bpm) or stress (155 ± 5 vs. 152 ± 8 bpm). PBV data are shown in the Table. Whereas HV showed no significant difference between the calculated rest and stress PBV (p=0.72), stress PBV tended to be higher than rest PBV in the Patients (p< 0.06). In both the HV and Patients, stress flows were significantly higher when compared to rest, and stress transit time was significantly shorter when compared to rest. When compared to HV, the patient stress flows were significantly lower and stress PTT was significantly longer.

Conclusion: In studies of healthy volunteers during exercise, the filling pressure of the LV does not increase to a great degree. In heart failure patients, however, there may be an increase in filling pressures during exercise. In the present study, we show that in healthy volunteers, exercise induced increases in pulmonary blood flow are well balanced by a reduction in PTT, resulting in no significant change in PBV. In the heart failure patients, however, a mismatch between increases in pulmonary flow and reductions in PTT may result in increased PBV. Further work is needed to explore the possibility that increases in PBV in heart failure patients during exercise may be predictive of increased LV filling pressures.

Table

	Rest Pulmonary Flow (ml/sec)	Rest Pulmonary Transit Time (sec)	Rest Pulmonary Volume (ml)	Exercise Pulmonary Flow (ml/sec)	Exercise Pulmonary Transit Time (sec)	Exercise Pulmonary Volume (ml)
Healthy Volunteers (n=10)	97 ± 6	7 ± 1	636 ± 66	181 ± 12 †	4 ± 0 †	668 ± 65
Patients (n=7)	79 ± 7	7 ± 1	571 ± 46	135 ± 15 †*	6 ± 0 †*	759 ± 88‡

Rest and Exercise Pulmonary Blood Flow, Pulmonary Transit Time, and calculated Pulmonary Volumes at Rest and Exercise in healthy volunteers and patients with heart failure.
Mean ± SEM
†p<0.05 Exercise vs. Rest
‡p<0.06 Exercise vs. Rest
*p<0.05 Patients vs. Healthy Volunteers

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Kiosk 10R-FB-09
Right Heart Remodeling in Pulmonary Hypertension Associated with Left Heart Disease and Chronic Thromboembolic Pulmonary Hypertension: Insights FBom Cardiac Magnetic Resonance

Blanca Domenech-Ximenos, MD, PhD[1], Bernardo Ayala, MD[2], Juan J Rodriguez-Arias, MD[3], Andrea Arenas, MD[2], Carmen Palacios, MD[2], Eduard Solé, MD[2], Susanna Prat-González, MD, PhD[2], Alessandro Affronti, MD, PhD[2], Ivan Vollmer, MD[1], Marcelo Sánchez, MD[1], Elena Sandoval, MD, PhD[2], Daniel Pereda, MD, PhD[2], Joan Albert Barberà, MD, PhD[4], Bárbara Vidal, MD, PhD[2], Isabel Blanco, MD, PhD[4], Manuel Castella, MD, PhD[2], Ana García-Alvarez, MD, PhD[2]

[1] *Centre de Diagnòstic per la Imatge, Hospital Clínic de Barcelona, Spain*
[2] *Institut Cardiovascular. Hospital Clínic de Barcelona. Spain*
[3] *University Health Network Toronto General Hospital*
[4] *Institut Clínic Respiratori. Hospital Clínic de Barcelona. Spain*

Background: Right ventricular (RV) and right atrial (RA) remodeling are common in the wide spectrum of diseases that produce pulmonary hypertension (PH) and are relevant prognostic factors for heart failure and mortality. The mechanisms associated with the differential RA and RV adaptation in different diseases causing PH are not completely understood. Our aim was to compare RA and RV remodeling in patients with PH associated with left heart disease (LHDPH - group 2) and chronic thromboembolic PH (CTEPH –group 4).

Methods: A total of 32 patients with PH were included: 17 with LHDPH and 15 with CTEPH eligible for pulmonary endarterectomy. All of them underwent, within 4 weeks, a right heart catheterization (RHC) and a contrast cardiac magnetic resonance (CMR) to assess cardiac dimensions, function, and tissue characterization. Echocardiography was also performed to evaluate the presence and severity of tricuspid regurgitation (TR), considering severe if grade >/= 3.

Results: Mean age was 66.7±13 years, 50% female. Patients with LHDPH were older (72.5±9.4 vs 60.3±15.1 years; p=0.009). As expected LHDPH patients exhibited higher pulmonary arterial wedge pressure (25.2±6.2 vs 8.2±1.9 mmHg, p< 0.001). There were no differences in mean pulmonary arterial pressure (40.3±9.0 vs 35.9±10.2 mmHg) and cardiac index (2.3±1.0 vs 2.2±0.4 L/