4890-A-2324 MODERATED POSTER   P-01

Noninvasive Pressure-Volume Loops using the Time-Varying Elastance Model and MRI – A Porcine Validation during Transient Preload Conditions

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Background. Pressure-volume (PV) loops have potential utility in heart failure evaluation but requires invasive measurements. Recently a noninvasive PV loop method was proposed. The method is based on a time-varying elastance model coupling pressure with left ventricular (LV) volume by MRI1-3 (Fig 1A). Elastance is scaled with a validated transfer function using brachial cuff pressures to estimate peak aortic pressure which, in the absence of aortic stenosis, is approximately equal to peak LV pressure4. LV end diastolic pressure (EDP) requires a user-estimation, an inherent limitation with limited impact on the PV loop1. Here, we expand the initial validation performed in 9 pigs with pressures and volumes measured sequentially, with simultaneous measurements concurrent to a preload reduction.

Methods. We performed PV loop experiments under 0.55T MRI-guidance in 15 pigs (n=7 naïve, n=8 six months post induction of ischemic cardiomyopathy). Simultaneously to a preload alteration by inferior vena cava (IVC) occlusion, we measured invasive LV and aortic pressures, and acquired a long-axis (LAX) real-time MR image (bSSFP, TE/TR/θ=1.4ms/77ms/80°, 2.3x2.3x8mm resolution, 360x270mm FOV, acceleration rate 3, 76ms temporal resolution). Centerline rotation of LAX segmentations were used to derive 3D volumes5. Invasive PV-loops were derived by combining pressure and volume signals6. Model-based PV loops were derived using real-time 3D volumes, peak aortic pressures and invasive LV-EDP as input. Stroke work, potential energy, energy efficiency, and peak elastance were quantified (Fig 1B).
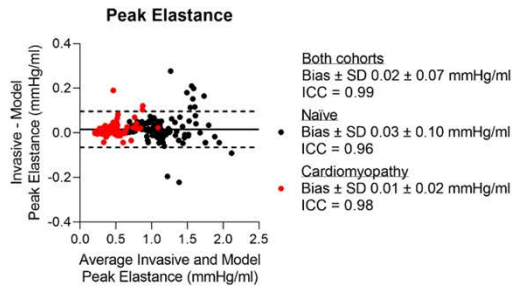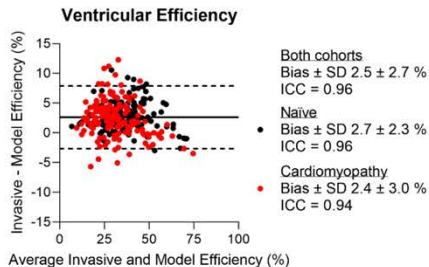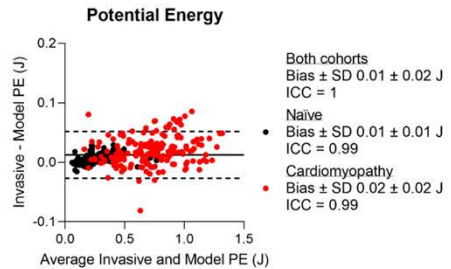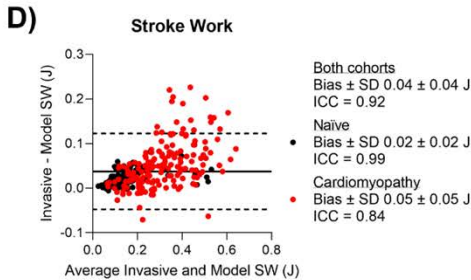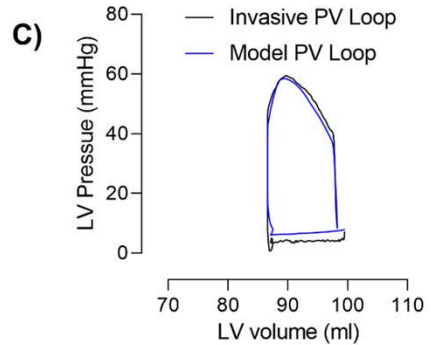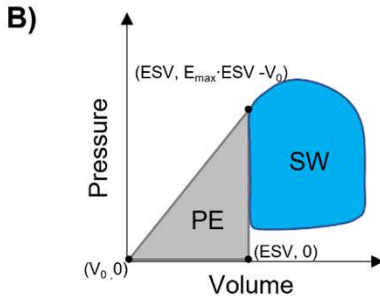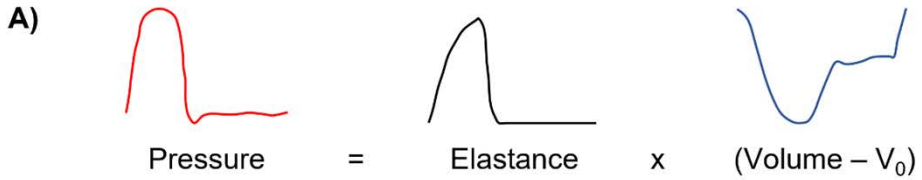
Results. We recorded a total of 330 PV loops, ~22 per pig experiment, and found an excellent agreement and low bias between the invasive and model-based stroke work (bias:0.04±0.04J), potential energy (bias:0.01±0.02J), energy efficiency (bias:2.5±2.7%) and peak elastance (bias:-0.02±0.07mmHg/ml) (Fig1CD).

Conclusion. An elastance model-based estimation of PV loops and associated hemodynamic parameters at transient loading conditions was validated. Further validation of the cuff to peak aortic pressure approximation4 and user-estimated LV-EDP are warranted.

References

1. Seemann et al. Circ CVI 2019 2. Sjöberg et al. Clin Physiol Funct Imag 2021 3. Suga et al. Circ 1973 4. Kelly JACC 1992 5. Seemann et al JCMR 2023 In press.

Figure 1. A) Schematic overview of noninvasive PV loops using the time-varying elastance model. B) Schematic PV loop illustration with associated hemodynamic parameters. The blue area within the PV loop correspond to the stroke work (SW). The grey triangular area defined by V0, peak elastance, and end systolic volume (ESV) approximates the potential energy (PE). Energy efficiency is calculated as SW/(SW+PE). C) Example of corresponding invasively measured and model-based PV loops. D) Bland-Altman plots of hemodynamic parameters. Black: naïve animals; Red: cardiomyopathy.

A)

Pressure = Elastance × (Volume − V_0)

B)

(ESV, $E_{max} \cdot$ESV −V_0)

Pressure

SW

PE

(V_0, 0)

(ESV, 0)

Volume

C)

— Invasive PV Loop
— Model PV Loop

LV Pressue (mmHg)

LV volume (ml)

D)

Stroke Work

Invasive − Model SW (J)

Average Invasive and Model SW (J)

Both cohorts
Bias ± SD 0.04 ± 0.04 J
ICC = 0.92

Naïve
Bias ± SD 0.02 ± 0.02 J
ICC = 0.99

Cardiomyopathy
Bias ± SD 0.05 ± 0.05 J
ICC = 0.84

Potential Energy

Invasive − Model PE (J)

Average Invasive and Model PE (J)

Both cohorts
Bias ± SD 0.01 ± 0.02 J
ICC = 1

Naïve
Bias ± SD 0.01 ± 0.01 J
ICC = 0.99

Cardiomyopathy
Bias ± SD 0.02 ± 0.02 J
ICC = 0.99

Ventricular Efficiency

Invasive − Model Efficiency (%)

Average Invasive and Model Efficiency (%)

Both cohorts
Bias ± SD 2.5 ± 2.7 %
ICC = 0.96

Naïve
Bias ± SD 2.7 ± 2.3 %
ICC = 0.96

Cardiomyopathy
Bias ± SD 2.4 ± 3.0 %
ICC = 0.94

Peak Elastance

Invasive − Model
Peak Elastance (mmHg/ml)

Average Invasive and Model
Peak Elastance (mmHg/ml)

Both cohorts
Bias ± SD 0.02 ± 0.07 mmHg/ml
ICC = 0.99

Naïve
Bias ± SD 0.03 ± 0.10 mmHg/ml
ICC = 0.96

Cardiomyopathy
Bias ± SD 0.01 ± 0.02 mmHg/ml
ICC = 0.98

Time-resolved dynamic lung water magnetic resonance imaging during exercise stress

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Background: Quantification of lung water dynamics during exercise stress is therefore of interest to unmask latent heart failure1. In this study, we develop a continuous 3D MRI method to quantify lung water dynamics between rest and exercise stress.
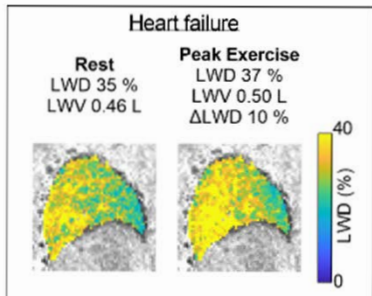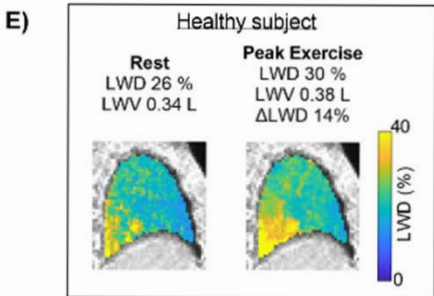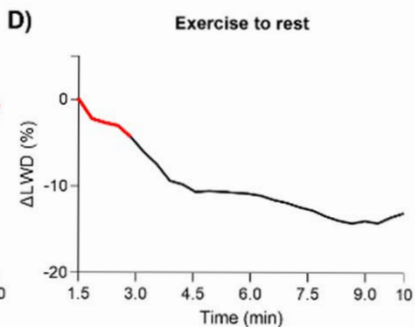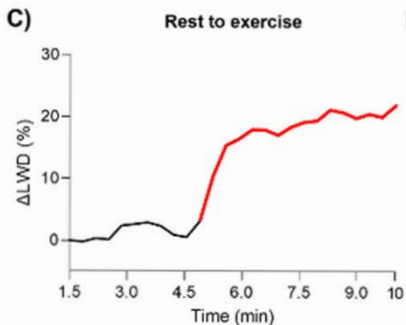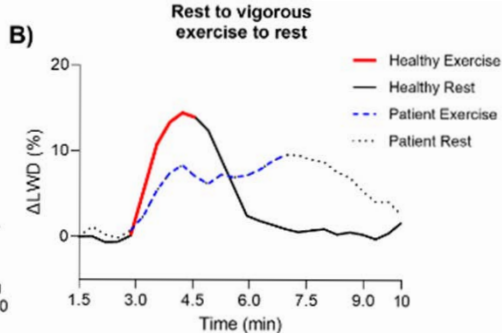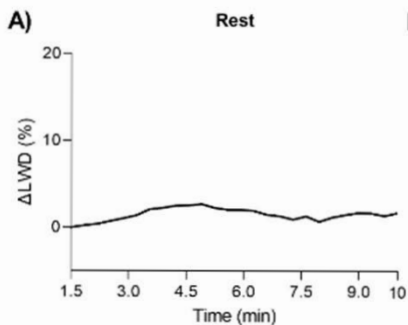
Methods: Free-breathing data was acquired at 0.55T2 using a continuous 3D stack-of-spirals proton density weighted MRI sequence with isotropic resolution3. Time-resolved images were derived using a motion compensated sliding-window reconstruction with 90s temporal resolution and 20s temporal increment. The method was evaluated in a porcine model of lung water accumulation (n=5), and in 15 healthy subjects and 2 patients with compensated heart failure imaged in transitions between rest and exercise stress using a supine MRI-compatible pedal ergometer. Time-resolved lung water density (LWD) and percent change in LWD (ΔLWD), and accumulation rates dLWD/dt were quantified using an automated pipeline4. The measured LWD were corroborated with cardiac output (CO) by phase contrast MRI. Pulmonary arterial wedge pressure (PAWP) was measured simultaneously to imaging in pigs.

Results: A ΔLWD increase of 3.3±1.5% was achieved in the porcine model, with a peak lung water accumulation rate of 1.01 ±0.81%/min. PAWP increased by 101±112%, p=0.009, and CO decreased by -28±15% (p=0.01), suggesting that measured ΔLWD was predominantly extravascular. Healthy subjects developed a ΔLWD of 7.8±5.0% during moderate exercise, peaked at 16±6.8% during vigorous exercise, and remained unchanged over 10-minutes at rest (-1.4±3.5%, p=0.18). CO increased from 4.5±1.5 L/min at rest to 7.5±2.6 L/min at moderate exercise, p=0.0004. Patient and healthy subject LWD were similar at rest (28±10% and 28 ±2.9%), respectively, as was peak vigorous ΔLWD (17±10% vs 16±6.8%) (Figure). Accumulation rates were slower in patients than in healthy subjects (2.0±0.1%/min vs 2.6±0.9%/min).

Conclusion: Dynamic changes in LWD can be quantified during exercise using a continuous 3D acquisition and a sliding-window, motion compensated image reconstruction.

References._1.Thompson_et_al_JCMR_2019; 2.Campbell-Washburn_et_al_Radiology_2019; 3.Javed_et_al_MRM2022; 4. Seemann_et_al_JCMR_2022.

Figure. Changes in lung water density (ΔLWD) in a heathy subject (solid line) and a patient with heart failure (dashed line) at rest (black) and exercise stress (red). A) ΔLWD at rest. B) ΔLWD starting at rest, followed by vigorous exercise targeting maximum heart rate, and then rest again. C) ΔLWD at rest, followed by exercise. D) Dynamic ΔLWD starting at moderate exercise then rest. E) LWD-maps in a sagittal slice at rest and after 10 minutes of moderate exercise in a healthy subject and a patient with heart failure. LWV, lung water volume.

A) Rest

B) Rest to vigorous exercise to rest

- Healthy Exercise
- Healthy Rest
- Patient Exercise
- Patient Rest

C) Rest to exercise

D) Exercise to rest

E)

Healthy subject

Rest
LWD 26 %
LWV 0.34 L

Peak Exercise
LWD 30 %
LWV 0.38 L
ΔLWD 14%

Heart failure

Rest
LWD 35 %
LWV 0.46 L

Peak Exercise
LWD 37 %
LWV 0.50 L
ΔLWD 10 %

Left ventricular underfilling leads to increased kinetic energy and contractility in patients with precapillary pulmonary hypertension

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The author has chosen not to publish the abstract

Athletes decrease right ventricular volumes more than sedentary individuals during exercise—an exercise real-time cardiac magnetic resonance imaging study

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Background: The athlete's heart has enlarged cardiac chambers with preserved systolic and diastolic function at resting conditions. However, there are conflicting results regarding the physiological mechanisms causing augmented cardiac output during exercise in athletes compared to sedentary controls. Therefore, the aim of this study was to investigate the mechanisms of increased cardiac output from rest to exercise in athletes and sedentary controls.

Methods: Twenty endurance-trained athletes (9 females) and 13 age and sex-matched sedentary healthy controls (6 females) underwent exercise cardiac magnetic resonance using a cycle ergometer. Images were acquired at rest and during steady-state moderate and vigorous intensities (defined as 60% and 80% of expected maximal heart rate). Left ventricular (LV) and right ventricular (RV) end-diastolic volume (EDV) and end-systolic volume (ESV) were measured at end-expiration. Differences were assessed by two-way mixed model ANOVA with Tukey post-hoc test and Bonferroni correction was used to adjust for multiple comparisons. $P < 0.05$ was considered statistically significant.

Results: Figures 1 and 2 show left and right ventricular volume changes in athletes and controls at rest and during moderate and vigorous exercise intensities. LVEDV decreased from rest to exercise at vigorous intensity by 8% in athletes and 9% in controls. Furthermore, LVESV decreased in athletes by 25% and in sedentary controls by 20%. The largest differences at vigorous intensity between groups were seen for the RV. Athletes decreased RVEDV by 20%, while RVEDV in controls was unchanged. RVESV in athletes decreased by 30%, while sedentary controls had unchanged RVESV ($p < 0.05$ for interaction effect between groups).

Conclusions: Athletes decrease RVESV more than sedentary controls, while the response of LVEDV and LVESV to exercise were not dependent on training status. These results may imply that the right ventricle act as a conduit in athletes, which may be a success factor for increasing cardiac output.

Athletes decrease right ventricular volumes more than sedentary individuals during exercise—an exercise real-time cardiac magnetic resonance imaging study

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Tables and Figures

Table 1. Subject characteristics at rest

	Athletes (n=20)	Controls (n=13)
Male / Female (n)	11 / 9	7 / 6
Age (years)	40 ± 10	40 ± 12
Height (cm)	172 ± 8	176 ± 5
Weight (kg)	67 ± 9	69 ± 7
BMI (kg/m^2)	22 ± 2	22 ± 2
BSA (m^2)	1.8 ± 1.5	1.8 ± 0.1
Heart rate (bpm)	59 ± 10	72 ± 12
SBP (mmHg)	123 ± 13	118 ± 16
DBP (mmHg)	75 ± 8	73 ± 10
Training volume (h/week)	7 ± 3 *	0 ± 1
VO$_2$peak (ml/kg/min)	54 ± 6 *	38 ± 7

* $p < 0.05$ athletes vs controls

Values denote frequencies or mean ± SD. BMI: body mass index; BSA: body surface area; DBP: diastolic blood pressure; SBP: systolic blood pressure; VO$_2$peak: peak oxygen consumption.

Athletes
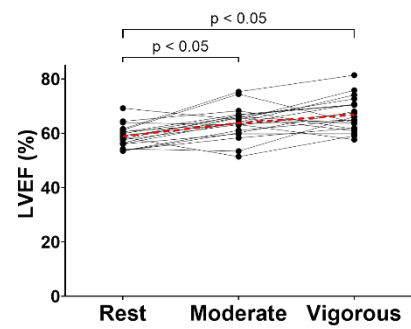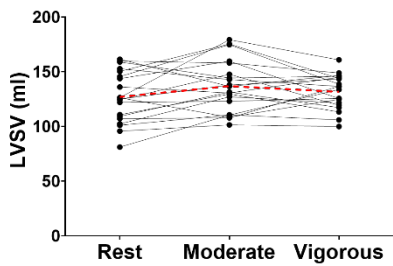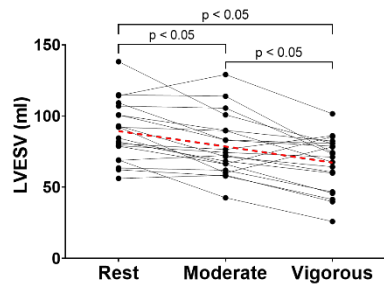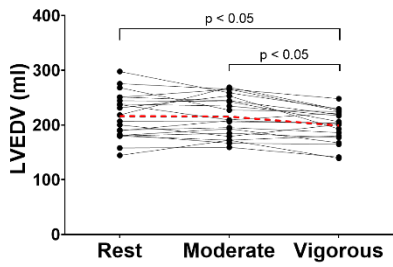
Sedentary

Figure 1. Left ventricular end-diastolic volume (LVEDV), end-systolic volume (LVESV), stroke volume (LVSV), and ejection fraction (LVEF) in athletes (left column) and sedentary controls (right column) at rest, during moderate and vigorous intensity exercise. The dashed lines denote mean values in athletes (red) and sedentary controls (blue).

Athletes

RVEDV (ml) — p < 0.05 (Rest–Moderate), p < 0.05 (Rest–Vigorous)

RVESV (ml) — p < 0.05 (Rest–Moderate), p < 0.05 (Rest–Vigorous)

RVSV (ml) — p < 0.05 (Rest–Vigorous), p < 0.05 (Moderate–Vigorous)

RVEF (%) — p < 0.05 (Rest–Moderate), p < 0.05 (Rest–Vigorous)

Sedentary

RVEDV (ml)

RVESV (ml)

RVSV (ml)

RVEF (%)
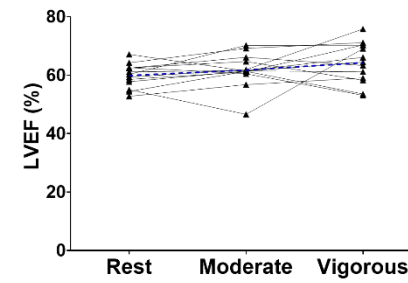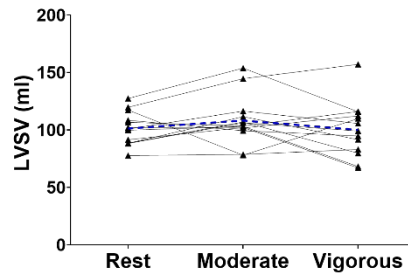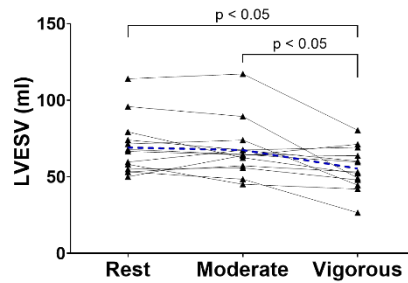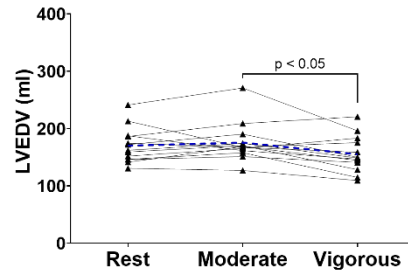
(x-axis categories: Rest, Moderate, Vigorous)
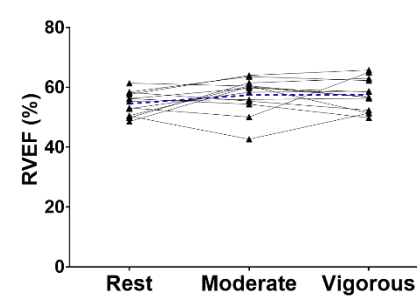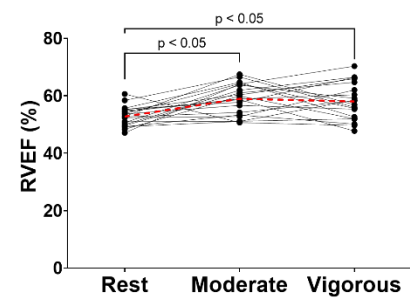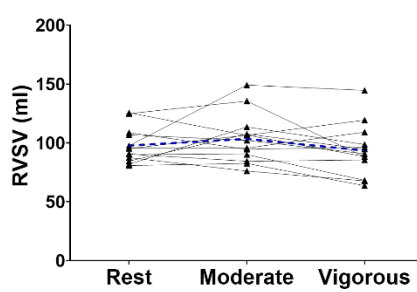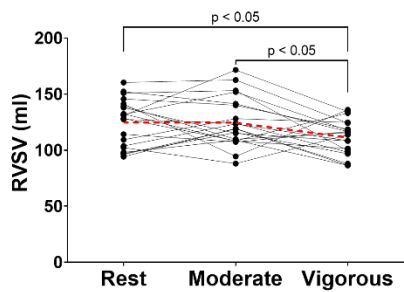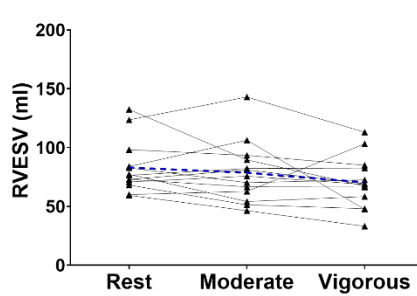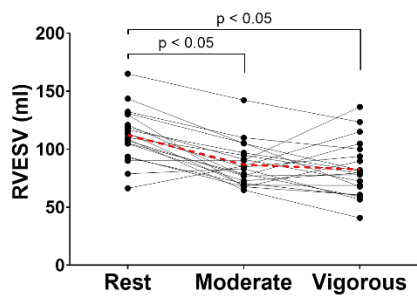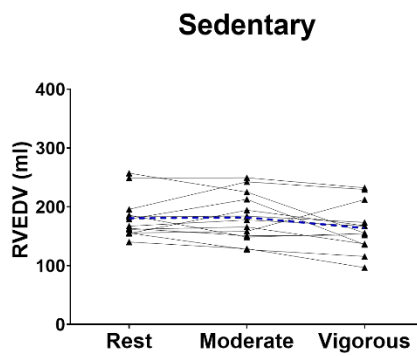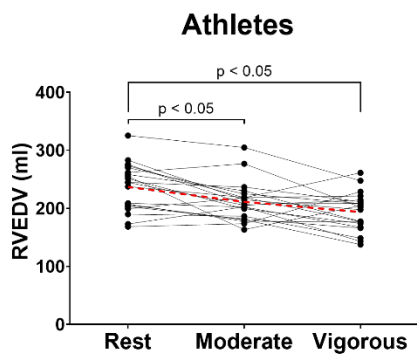
Figure 2. Right ventricular end-diastolic volume (RVEDV), end-systolic volume (RVESV), stroke volume (RVSV), and ejection fraction (RVEF) in athletes (left column) and sedentary controls (right column) at rest, during moderate and vigorous intensity exercise. The dashed lines denote mean values in athletes (red) and sedentary controls (blue).

4855-A-2301 MODERATED POSTER   P-05

In Fontan patients, the amount of liver blood flow to the lungs has a gradual effect on pulmonary shunting and oxygen saturation at rest

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The author has chosen not to publidh the abstract.

Cardiovascular magnetic resonance derived pressure volume loop variables in patients with ST-elevation myocardial infarction provide physiological information beyond ejection fraction

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The author has chosen not to publidh the abstract.

Right ventricular dyssynchrony assessed in multiple MR views predicts outcome in pulmonary arterial hypertension

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The author has chosen not to publidh the abstract.

Automated Detection of Cardiac Rest Period for Trigger Delay Calculation for iNAV-based Coronary Magnetic Resonance Angiography

Gregory Wood[1,2], Alexandra Uglebjerg Pedersen[1,2], Karl P. Kunze[3,4], Radhouene Neji[3,4], Reza Hajhosseiny[4,5], Jens Wetzl[6], Seung Su Yoon[6], Michaela Schmidt[6], Bjarne Linde Nørgaard[1,2], Claudia Prieto[4,7,8], René M. Botnar[4,7,8,9,10]

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The author has chosen not to publidh the abstract.

Left atrial strain in a large healthy population

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Background

Echocardiographic evaluation of the left atrium (LA) has recently been proposed to include LA conduit strain, a direct measurement of LA myocardial function during the passive phase of left ventricular (LV) filling. Since passive LV filling is driven by suction of blood from the LA, and both LA and LV strain decline with increasing age, we hypothesize that variations in age and LV global longitudinal strain (LV GLS) explains most of LA conduit strain.

Materials and methods

Echocardiographic acquisitions and analyses were performed according to current recommendations at a European Association of Cardiovascular Imaging (EACVI) accredited echocardiographic laboratory. Strain measurements were performed by EACVI certified cardiologists with >15 years of experience with strain imaging, using dedicated software (GE HealthCare EchoPAC). The associations of LA conduit strain as outcome variable with age and LV GLS was assessed by multiple linear regression analysis.

Results

In total 1412 subjects (56% females) with mean (SD) age 57 (12) years were included. Mean LVEF was 60.4% in females and 59.6% in males. Totally, 54% of the variance in LA conduit strain between subjects was explained by age and LV GLS (Table 1). Both characteristics were positively associated with LA conduit strain, indicating lower absolute LA conduit strain with higher age and lower absolute LV GLS. Three years of higher age had a similar effect as one percent lower absolute LV GLS.

Conclusion

In this large healthy population, most of the variance in LA conduit strain between subjects was explained by age and LV GLS. Increasing age had approximately twice the effect on the absolute decrease of LA conduit strain as decreasing LV GLS had. In conclusion, both age and LV function should be considered when interpreting LA conduit strain.

Table 1. LA conduit strain by age and LV GLS.

Characteristic	Standardized Model		Original Model		
	Beta	95% CI[1]	Beta	95% CI[1]	p-value
Age	0.59	0.55, 0.63	0.33	0.31, 0.35	<0.001
Left ventricular global longitudinal strain	0.29	0.25, 0.32	0.97	0.83, 1.1	<0.001
R^2	0.536				
[1] CI = Confidence Interval					

A

$R^2 = 0.47, P < 0.001, n = 1332$

B

$R^2 = 0.22, P < 0.001, n = 1315$
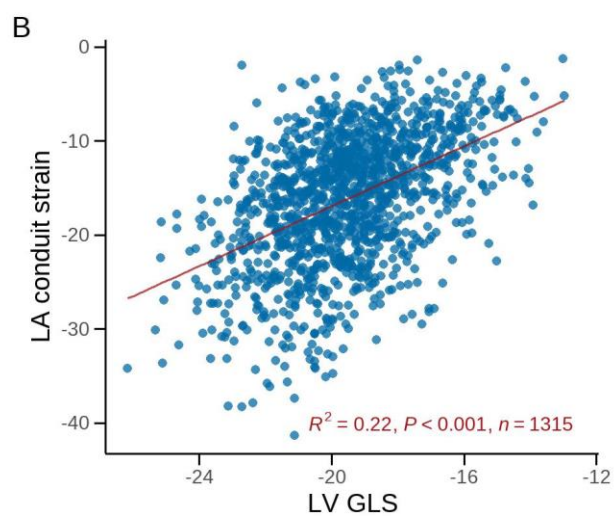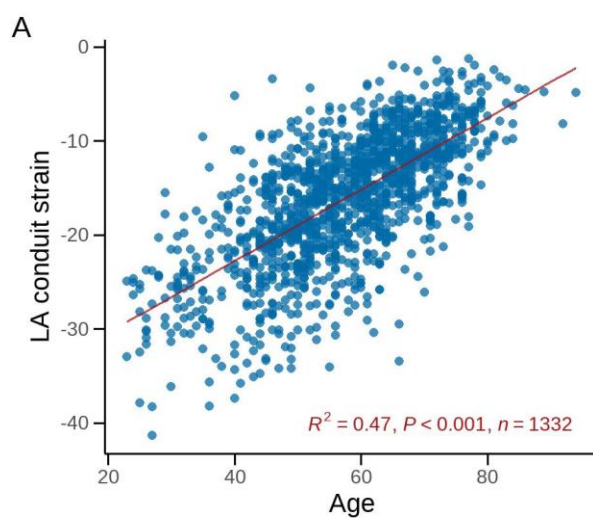
Pulmonary blood volume across heart failure phenotypes

Anders Nelsson[1,2], Per Arvidsson[1,2], Jonathan Edlund[1,2], Martin Magnusson[1,2], Gustav Smith[1,2,3,4], Katarina Steding-Ehrenborg[1,2], Håkan Arheden[1,2]

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[4] Göteborgs Universitet

The author has chosen not to publidh the abstract.

4758-A-2301   P-11

Fetal 3D cine cardiovascular MRI: Improved image quality with region-optimized virtual coils

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The author has chosen not to publish the abstract.

4762-A-2301   P-12

Hemodynamic forces predict left ventricular remodeling following cardiac resynchronization therapy in heart failure with left bundle branch block

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The author has chosen not to publidh the abstract.

Left ventricular haemodynamic phenotyping using 4D flow CMR detect subclinical abnormalities in non-obstructive hypertrophic cardiomyopathy

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The author has chosen not to publish the abstract.

4774-A-2301   P-14

Diagnostic confidence with quantitative CMR perfusion mapping increases with increased coverage of the left ventricle

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The author has chosen not o publish the abstract.

Free-breathing mSASHA extracellular volume in patients is significantly lower than MOLLI in accordance with previous experimental data

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The author has chosen not to publidh the abstract.

Echocardiographic indices of right ventricular function differentiate pre- and post-capillary pulmonary hypertension

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Background: The importance of assessing right ventricular (RV) function in heart failure is well established. Methods evaluating intrinsic RV function using Doppler echocardiography estimating RV -pulmonary coupling assessment might improve the understanding of RV response due to increase in RV afterload in different conditions.

Aim: To investigate RV function and RV-pulmonary coupling in patients with pre- and postcapillary pulmonary hypertension

Material and Methods: We investigated 85 patients with pulmonary hypertension (PH) defined as mPAP >20mmHg from right heart catheterization (RHC). Those having PH with PCWP≤15mmHg and PVR ≥ 3WU were categorized as pre-capillary PH (preCPH, n=60) and those with PCWP>15mmHg and PVR <3WU as post-capillary PH (postCPH, n=25). Of those 25 having postCPH, 16 had HFrEF (LVEF<50%) and 9 HFpEF)LVEF>=50%). Doppler echocardiography and RHC were done simultaneously.

Results: Pulmonary artery acceleration time (<0.01), sPAP (<0.01) and regional RV strain (p<0.05) differed between pre- and postCPH, whereas RV -pulmonary coupling did not, table 1. The indirect echocardiographic measure demonstrating strongest association with Ees/Ea (volume method= SVrhc/ESVecho) in patients with preCPH was mPAP/CO (r=0.61, fig 1A), RV s'/sPAP and RVGLS/sPAP (r=0.62, fig 1B, and 0.57 respectively). RV e', RV segmental and global strain were lower in HFrEF compared with HFpEF but RV-pulmonary coupling did not differ between groups.

Conclusion: Right ventricular-pulmonary coupling and RV free wall strain had low accuracy to differentiate pre- from post CPH which was not the case for PAAt, sPAP and RV regional strain. mPAP/CO and RVs'/sPAP suggests promise as markers for right ventricular- pulmonary coupling, especially in pre CPH.

Table 1.

	Pre-CPH n=60	Post-CPH (all) n=25	Post-CPH (HFrEF) n=16	Post-CPH (HFpEF) n=9	Anova Kruskal Wallis
Age (years)	66 (12)	66 (12)	65 (13)	68 (10)	ns
BSA (m^2)	1.8 (0.2)	2.0 (0.3)	2.0 (0.3)	1.9 (0.3)	ns
AF (%)	27	36	50	44	ns
Females (%)	65	56	50	67	0.006
RV function and hemodynamics (ECHO)					
PAAt (ms)	75 (20)	91 (21)*	84(17)	99 (25)	0.011
sPAP (mmHg)	62 (20)	50 (15(*	48 (10)	58 (19)	0.041
SV (ml)	67 (19)	87 (40)	61 (26)	80 (19)	ns
mPAP/CO (mmHg/L/min)	10 (6)	7 (3)	8 (3)	7 (2)	ns
CO (L/min)	4.6 (1.4)	5.1 (2.0)	4.4 (1.5)	6.4 (2.1)	0.009
TDI RVs' (cm/s)	9.1 (2.8)	8.6 (2.8)	8.0 (2.3)	9.5 (3.4)	ns
TDI RVs' <10 cm/s (%)	64	65			
TDI RVe' (cm/s)	8.9 (4.5)	10.6 (3.5)	9.1(1.7)	12.0(4.5)	ns
STE regional systolic strain, mid free wall (%)	-14 (9)	-18 (7)*	-15(6)	-22(5)*	0.016
STE RV GLS (%)	-14 (7)	-16 (6)	-14(5)	-20(6)*	
STE RV free wall GLS >-19 % (%)	71	53			
TDI RVs'/sPAP (cm/s/mmHg)	0.17 (0.08)	0.17 (0.05)	0.16 (0.05)	0.17 (0.06)	ns
RV GLS/sPAP (%/mmHg)	0.64 (0.34)	0.76 (0.27)	0.70 (0.27)	0.80 (0.23)	ns
Right heart catheterisation					
sPAP (mmHg)	60 (17)	52 (12)*	33 (6)	36 (7)	ns
RVEDP (mmHg)	9 (4)	13 (4)**	11(4)	16 (4)*	<0.001
RV Ees/Ea (volume method)	0.6 (0.6)	0.8 (0.6)	0.6 (0.6)	0.3 (0.1)	

Data presented as mean (SD)

*=<0.01 **<0.001

Non-parametric T-test (Mann-Whitney) comparing Pre and Post CPH as well as PostCPH type HFrEF and HFpEF

Kruskal Wallis variance analysis comparing PreCPAH vs PostCPH type HFrEF and HFpEF. RV Ees/Ea volume method, SVrhc/ESVecho

4792-A-2301   P-16

Patients with post–acute sequelae of Covid-19 presenting as postural orthostatic tachycardia syndrome have lower native T1 stress reactivity during long-term follow-up

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Background: Post-acute sequelae of Covid-19 (PASC) is a novel clinical syndrome with diverse symptoms. It has previously been reported that PASC patients can develop postural orthostatic tachycardia syndrome (POTS) and that Covid-19 induce microvascular endothelial dysfunction in acutely ill, hospitalized patients, that persists post discharge. Whether myocardial inflammation and reduced stress reactivity in the microcirculation contributes to POTS pathophysiology in PASC remains unclear. Myocardial inflammation can be detected by cardiovascular magnetic resonance (CMR) imaging using native T1 mapping. Furthermore, native T1 has previously been shown to detect the change in myocardial perfusion during adenosine stress in normal physiology. Therefore, this study aimed to elucidate differences in native T1 and stress reactivity (deltaT1) between patients with PASC, both with and without POTS and healthy subjects.

Methods: Patients with PASC due to prior mild Covid-19 infection (n=26, mean ± SD age 41 ± 11 years, 92 % females) and healthy subjects (n=21, age 37 ± 15 years, 100 % females, p=0.08) underwent 1.5T or 3T CMR (MAGNETOM Aera, or Skyra, Siemens Healthcare, Erlangen, Germany) at least 6 months after the primary Covid-19 infection. 15 of 26 patients with PASC (39 ± 11 years, 87 % females) were diagnosed with POTS. Patients with PASC had no previous cardiac disease or notable comorbidities and were followed in outpatient care for long-term Covid-19 at Karolinska University Hospital, Stockholm Sweden. Native T1 maps were acquired with a prototype 5s(3s)3s modified look-locker inversion recovery sequence before and during adenosine stress. Global native T1 were acquired by carefully delineating endo- and epicardial borders. Delta T1 (%) was calculated as (stress-rest) /rest (%) for native T1.

Results: Patients with PASC had lower delta T1 compared with healthy subjects (3.7 ± 2.4 vs 5.5 ± 2.9 %, p=0.03, Figure 1). Moreover, patients with PASC POTS had a lower delta T1 than healthy subjects (3.2 ± 2.7 vs 5.5 ± 2.9 %, p=0.02, Figure 2), however there was no difference in delta T1 in patients with PASC POTS compared to patients with PASC non-POTS (3.2 ± 2.7 vs 4.3 ± 1.8, p=0.23, Figure 2).

There was no difference in native T1 at rest or stress in patients with PASC compared with healthy subjects, neither at 1.5 nor 3 T (p>0.05 for all). This remained true for all subgroup analysis of PASC POTS and PASC non-POTS.

Conclusion: Patients with PASC and particularly those with PASC POTS have lower native T1 stress reactivity compared with healthy subjects. These findings suggest presence of microvascular dysfunction as a pathophysiological mechanism in patients with PASC POTS long after initial infection and can be detected using non-contrast stress imaging.

Figure 1. Difference in delta T1 between healthy subjects and patients with PASC.
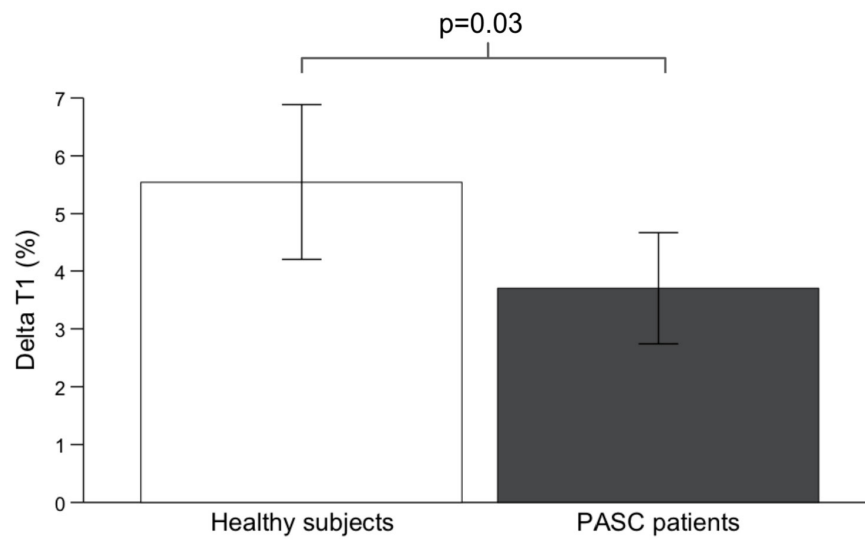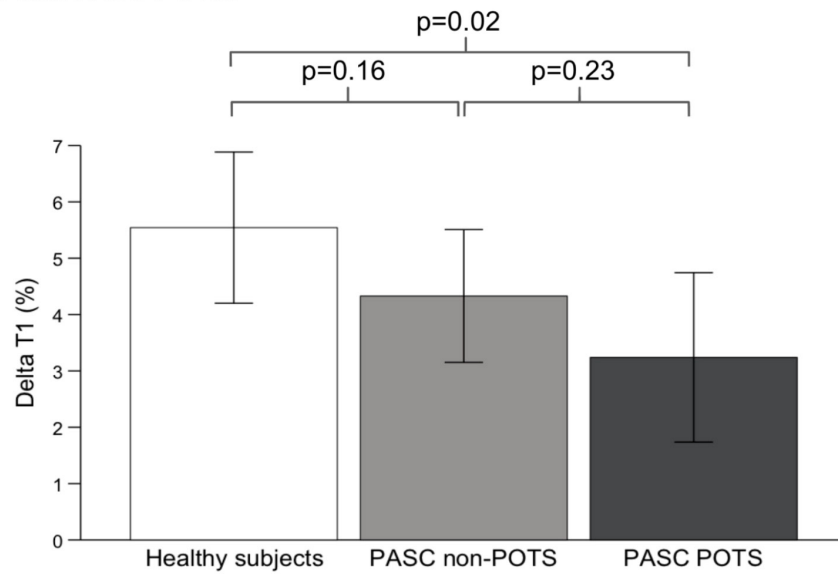

Figure 2. Differences in delta T1 between healthy subjects, patients with PASC non-POTS and patients with PASC POTS.

Alcohol septal ablation in patients with hypertrophic obstructive cardiomyopathy - does sex or initial septal thickness affect outcomes at one-year follow-up?

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Background

Alcohol septal ablation (ASA), a minimally invasive procedure to decrease the septal thickness and reduce the left ventricular outflow tract obstruction (LVOTO) is known to be an efficient treatment option for hypertrophic obstructive cardiomyopathy (HOCM). However, the influence of sex or initial basal ventricular septal thickness on outcomes after ASA remain unknown.

Methods

In total, 154 patients (50% females) that underwent ASA on a clinical basis at a Swedish university hospital between 2009-2021 were retrospectively included. Echocardiography was performed at baseline and 12 months after ASA. Heart catheterization recordings of pressures in the left ventricle and ascending aorta was performed, with peak-to-peak pressure gradients derived across the LVOT, at baseline and immediately after ASA. Successful outcomes were quantified with respect to remaining LVOTO after ASA, defined as LVOT pressure gradients <30 mmHg at rest, or <50 mmHg during Valsalva maneuver. Further, outcomes were quantified with respect to the need for re-intervention (ASA or myectomy) or need for pacemaker or implantable cardioverter defibrillator (ICD) at 1-year post ASA.

Results

Out of the complete cohort, 14% exhibited a remaining elevated LVOT pressure gradient after ASA, and 31% required either re-intervention (9%), pacemaker (19%) or ICD (3%). There were no significant sex differences in outcomes regarding LVOTO ($p=0.482$), need for re-intervention ($p=0.375$), post operative pacemaker ($p=0.806$) or ICD ($p=0.641$), respectively. However, there was a significantly higher rate of LVOTO in patients with septal thickness >23 mm ($p=0.004$), although, no significant correlation between septal thickness and need for re-intervention after ASA was observed ($p=0.308$).

Conclusion

Patients with septal thickness >23 mm were more likely to have remaining LVOTO after ASA, suggesting extended clinical evaluation and analysis of optimal treatment strategies for these patients regarding LVOTO-associated symptoms. Sex did not influence the outcomes after ASA, although extended assessment might be warranted in future studies.

4806-A-2301   P-18

LAMPOON prior to transcatheter valve-in-ring treatment of severe mitral regurgitation after mitral valve repair

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History of presentation

A 70-year old man presented with hemolytic anemia, dyspnea and decompensated heart failure one month after mitral valve repair with artificial chordae and mitral annuloplasty ring and implantation of a biological aortic valve prosthesis.

Investigations

Transesophageal echocardiography (TOE) revealed severe mitral regurgitation with prolapse of the P2 segment with flail. The anterior mitral leaflet had systolic anterior motion (SAM) with severe left ventricle outflow tract obstruction (LVOTO).

Cardiac computed tomography (CT) was used to assess the neo LVOT with a virtual 29 mm Edwards Sapien3 prosthesis in mitral position.

Management

The patient was accepted for a transcatheter valve-in-ring procedure with prior LAMPOON (tip-to-base) procedure (laceration of the anterior mitral leaflet to prevent outflow obstruction) to prevent further LVOTO.

A Swan-Ganz catheter with inflated balloon was inserted to the ascending aorta and a 6 French guide catheter with a snare from the left femoral artery. From the Swan-Ganz catheter, a 300 cm Astato PCI-leader was inserted, fetched by the snare in the descending aorta, and externalized. A "flying V" was created by a 6 French guide catheter JR4 with a piggyback catheter from the venous access. The "flying V" was positioned towards the anterior mitral valve leaflet that was lacerated using electrocautery (Fig 1). The patient became hemodynamically unstable, ECMO was initiated. An Edwards Sapien3 valve was deployed in the mitral ring. A True balloon was inflated between the Sapien valve and the LVOT to further reduce the LVOTO. After valve deployment, ECMO could be discontinued.

Follow-up

At 3-month-follow-up, the patient had less LVOTO, decreasing degree of hemolysis, no symptoms of heart failure or anemia.

Conclusion

We describe a successful LAMPOON procedure prior to valve-in-ring Edwards Sapien mitral prosthesis due to severe mitral regurgitation and LVOTO after mitral valve repair.

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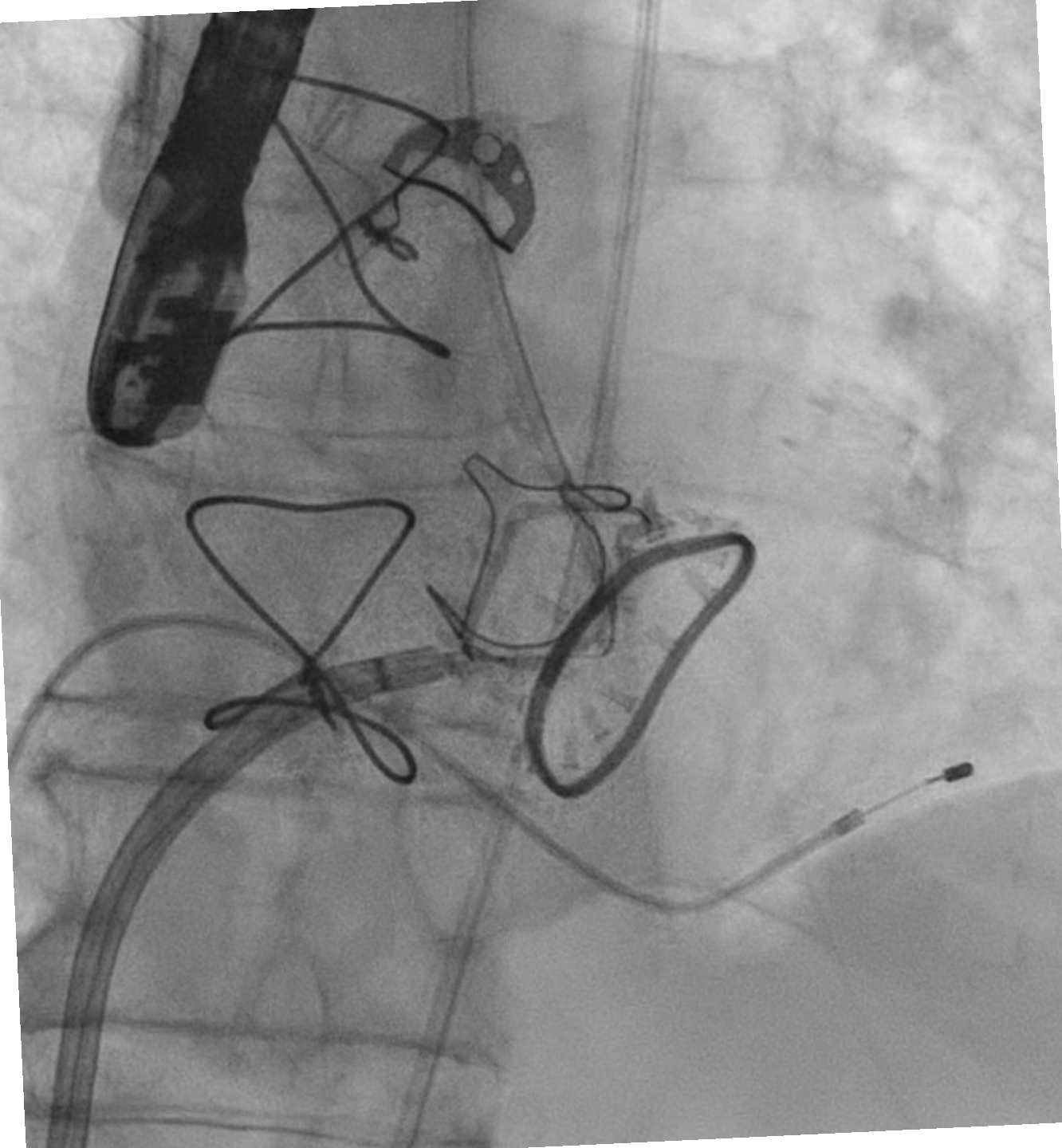
Lower left atrial function in adolescents and young adults with type 1 diabetes mellitus compared to healthy controls - an echocardiographic study

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The author has chosen not to publidh the abstract.

Nativa T1- och T2-kartor under stress kan inte skilja mellan patienter med hjärtinfarkt med normala kranskärl och kontroller

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Bakgrund:

Hjärtinfarkt med normala kranskärl (MINOCA), är ett akut koronart syndrom som vid klassisk hjärtinfarkt men utan förträngningar av kranskärlen på angiografi. Koronar mikrovaskulär dysfunktion (CMD) kan vara ett delförklaring i MINOCA. Magnetresonanstomografi av hjärtat (CMR) kan generera kvantitativa perfusionskartor, vilka kan användas till funktionell bedömning av mikrocirkulationens anpassningsförmåga till fysiologisk stress. Nativ T1- och T2-avbilning har tidigare visat sig spegla den koronara cirkulationens funktion under farmakologisk stress utan kontrastmedel. Det är dock oklart hur nativ T1 och T2 stämmer överens med kvantitativ perfusion hos patienter med MINOCA. Arbetet ämnar att utröna huruvida nativ T1- och T2-avbildning kan upptäcka störningar i mikrocirculationen hos MINOCA-patienter.

Material och metoder:

MINOCA- patienter från tidigare studier med normala CMR-fynd genomgick uppföljande CMR-avbildning i vila och under adenosininducerad stress. Nativa T1- och T2- kartor, och kvantitativa perfusionskartor under adenosinstress och i vila efter administrering av gadoterinsyra inhämtades. Perfusionsreserven (MPR) beräknades som perfusion i stress delat med vila.

Resultat:

15 patienter (59±7 år, 60% kvinnor) och 15 ålders- och könsmatchade kontroller undersöktes. Det förelåg inga skillnader i globala nativa T1- och T2- värden mellan grupperna. MINOCA-patienter hade lägre perfusion i stress jämfört med kontroller. Δ T1 korrelerade med MPR hos sammanvägda patienter och kontroller (p<0.05 för samtliga).

Konklusion:

Det fanns ingen skillnad mellan nativa T1- och T2-värden mellan MINOCA- patienter och friska kontroller, trots att perfusionen i stress var lägre hos MINOCA-patienter.

4823-A-2301   P-21

Right ventricular diastolic kinetic energy is increased in patients with precapillary pulmonary hypertension

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The author has chosen not to publidh the abstract.

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4824-A-2301   P-22

Diastolic filling in patients after heart transplantation is Impaired by an altered geometrical relationship between the left atrium and ventricle

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The author has chosen not to publidh the abstract.

4827-A-2301   P-23

Smaller ventricles but preserved cardiac pumping in adolescents born very preterm

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The author has chosen not to publidh the abstract.

Myocardial perfusion reserve in remote myocardium decreases significantly in patients with chronic coronary syndrome undergoing revascularization

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The author has chosen not to publidh the abstract.

Left ventricular systolic dysfunction after acute myocardial infarction is associated with a high symptom burden and worse secondary prevention

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The mortality and morbidity burden remain high for patients with LV dysfunction after acute myocardial infarction. There is a lack of contemporary data describing patients with HF post-AMI in terms of symptom burden and secondary prevention measures. The purpose was to describe patients with different degree of LV dysfunction after MI, their symptom burden, quality of life and adherence to secondary prevention measures.

We included adults, registered in the SWEDEHEART registry (2011-2018), diagnosed with AMI, with no previous HF. Stratified by degree of LV systolic impairment; EF >50%, EF 40-49%, EF 30-39% and <30%, assessed by echocardiography. Assessed at 6-10 weeks and 10-14 months after MI. Descriptive analyses were used for demographics and characteristics. Uni- and multivariable regression analyses was used to examine the associations between LV impairment and symptoms, occupation/working status, readmissions, quality of life (Eq5D), attending cardiac rehabilitation and physical training.

Patients with impaired LVEF would more often experience shortness of breath (5.6% vs 32.3% for EF < 30 vs EF >50 at second follow up) (figure). Patients with EF <30% had a lower Eq5D mean at first follow up than patients with normal LVEF (0.77 vs 0.82), smaller at second follow-up (0.80 vs 0.83). Subjects with EF <30% participated in heart rehabilitation to a lesser extent than those with normal EF, both at the first (28.7% vs 37.4%) as well as the second follow up (44.9% vs 55.5%). In multivariate regression analyses (table), a lower EF was associated with shortness of breath (OR = 7.47, 95% CI = 6.24-8.94), lower probability of working/studying (OR = 0.25, 95% CI = 0.20 – 0.31). Lower EF was associated with lower probability of participating in heart school (OR = 0,67, 95% CI = 0.58-0.68) and physical training (OR = 0.54, 95% CI = 0.46-0.63).

LV systolic dysfunction after MI is associated with high symptom burden and worse secondary prevention. This may indicate heart rehabilitation programs should be better adapted to this large population with great needs.

Table: Association between ejection fraction and different outcomes at 1 year adjusting for differences in risk factors and comorbidities. Each line shows the odds ratio [95% confidence interval] for each LVEF group.

Chest Pain

(50%-)	ref
(40-49%)	1.01 [0.83;1.21]
(30-39%)	1.11 [0.87;1.42]
(<30%)	0.76 [0.45;1.28]

Shortness of Breath

(50%-)	ref
(40-49%)	1.86 [1.66;2.08]
(30-39%)	4.08 [3.61;4.60]
(<30%)	7.47 [6.24;8.94]

Working or studying

(50%-)	ref
(40-49%)	0.90 [0.80;1.02]
(30-39%)	0.66 [0.56;0.77]
(<30%)	0.42 [0.31;0.56]

Sick Leave

(50%-)	ref
(40-49%)	1.07 [0.92;1.24]
(30-39%)	1.51 [1.26;1.82]
(<30%)	2.52 [1.90;3.35]

Smoking

(50%-)	ref
(40-49%)	1.04 [0.93;1.16]
(30-39%)	0.90 [0.77;1.04]
(<30%)	0.79 [0.62;1.02]

Snuffing

(50%-)	ref
(40-49%)	0.91 [0.82;1.01]
(30-39%)	0.97 [0.84;1.11]
(<30%)	0.76 [0.58;1.01]

Heart school Attendance

(50%-)	ref
(40-49%)	0.99 [0.93;1.05]
(30-39%)	0.84 [0.77;0.91]
(<30%)	0.68 [0.58;0.79]

Physiotherapy Attendance

(50%-)	ref
(40-49%)	0.97 [0.92;1.03]
(30-39%)	0.86 [0.79;0.94]
(<30%)	0.66 [0.57;0.77]

Any readmission

(50%-)	ref
(40-49%)	1.14 [1.07;1.22]
(30-39%)	1.37 [1.26;1.50]
(<30%)	1.88 [1.61;2.19]

MI readmission

(50%-)	ref
(40-49%)	1.07 [0.86;1.33]
(30-39%)	1.21 [0.91;1.60]
(<30%)	1.34 [0.83;2.16]

Figure
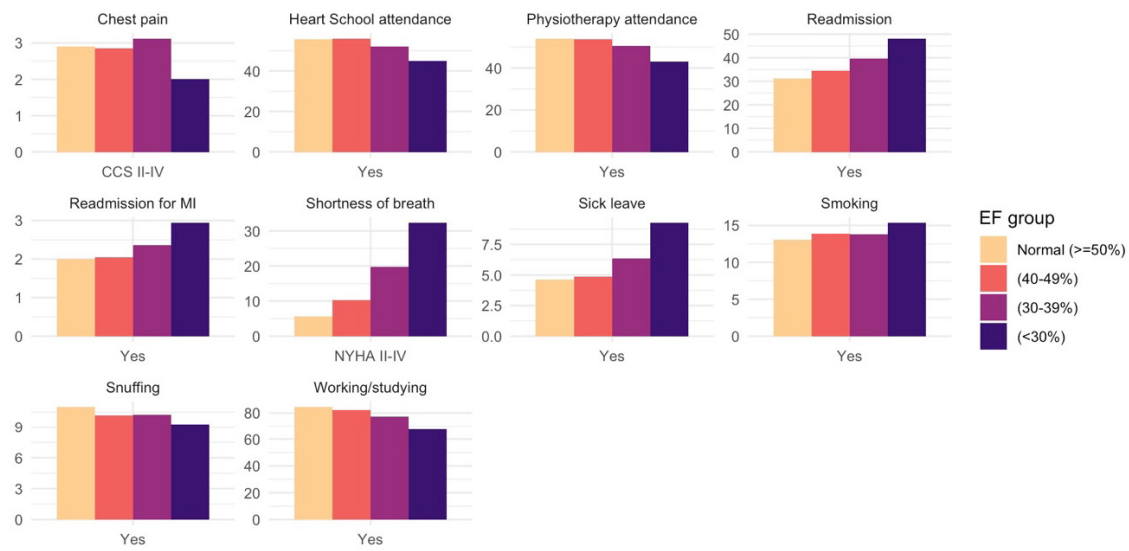
Plot describing recorded follow-up data at second follow-up visits. Each bar represents the
EF group percentage.

Exercise ECG and myocardial perfusion SPECT show high prognostic value up to five years after examination

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Background

The prognosis for patients with normal myocardial perfusion SPECT (MPS) or exercise ECG (Ex-ECG) is known to be favorable the first years after the examination. However, the long-term prognostic value of stress testing could primarily depend on risk factors such as age, gender, diabetes, and ischemic heart disease (IHD). The aim of this study was to compare the prognostic value of MPS and Ex-ECG for two different follow up periods (0-5 years and after 5 years) in relation to known risk factors.

Methods

908 patients (age 63 years, 49% male, 45% IHD) referred for MPS and Ex-ECG were prospectively included. Follow up was divided into two periods: 0-5 years and after 5 years. All subjects, except for deceased (n=30), were eligible for follow up after 5 years regardless of events during the first 5 years. The outcome was a composite of acute myocardial infarction, unstable angina, unplanned revascularization, and cardiovascular death. Kaplan-Meier curves and Cox proportional hazard analysis were used for risk assessment during follow up.

Results

The composite end point was reached in 95 patients (0-5 years follow up) and in 111 patients (after 5 years follow up) and the risk of event was primarily increased for the former (Figure 1). In multivariable models the strong predictive value of stress testing found for 0-5 years follow up (hazard ratio for MPS=3.0, CI=1.9-4.6 and Ex-ECG=2.1, CI=1.4-3.4) was lost after 5 years follow up (hazard ration for MPS=0.9 (CI=0.6-1.5) and Ex-ECG=1.0 (CI=0.6-1.5). Age, diabetes and reduced exercise capacity became important risk factors for follow up after 5 years, while male sex and IHD were significant predictors for both follow up periods (Table1).

Conclusions

MPS and Ex-ECG results have prognostic value for follow up duration less than five years but not after that. Long-term prognosis is primarily governed by underlying risk factors such as age, gender, diabetes, IHD and exercise capacity.

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Functional capacity is only to a small extent determined by right ventricular function in precapillary pulmonary hypertension.

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Background:

Right ventricular (RV) failure is a major determinant of outcome in precapillary pulmonary hypertension (PHpre-cap) and decreased exercise capacity is a marker of increased risk for death or lung transplantation. RV function can be assessed non-invasively by cardiac magnetic resonance imaging (CMR) or echocardiography (echo). New CMR measures of RV function, beyond RV ejection fraction (RVEFCMR), have emerged but it is unknown if these are associated with functional capacity, i.e. 6-minutes walking test (6MWT). The aim was to evaluate if RV functional measures by CMR and their echocardiographic equivalents are determinants of 6MWT in PHpre-cap patients.

Methods:

Forty-nine patients (57% women, mean age 61±16 years) with PHpre-cap (pulmonary arterial hypertension (n=40) and chronic thromboembolic PH (n=9)) underwent prospective assessment of RV function at rest with CMR and echo. From CMR, RVEFCMR, RV atrio-ventricular plane displacement (RVAVPD), maximum emptying velocity (S'CMR), RV fractional area change (RVFACCMR) and RV free wall longitudinal strain (RVFWSCMR) were measured. Equivalently echocardiographic measures of TAPSE, S´echo, RVFACecho and RVFWSecho. were assessed. 6MWT was performed in conjunction with the exams. Coefficients of determination (R2) was computed.

Results

Mean distance walked during 6MWT was 311±137 m (normal values 400-700m). RV functional impairment was shown for RVEFCMR, RVFWSecho and RVFACecho. 6MWT was determined of RVlatAVPD, RVFWSecho, RVFWSCMR, RVEFCMR, S'CMR and TAPSE by 11-23% (Table 1), while S'echo and RVFACCMR and RVFACecho did not determine functional capacity.

Conclusion

6MWT was markedly decreased in patient with PHpre-cap. However, functional capacity was only determined by a minor part of RVlatAVPD, TAPSE, RVEFCMR and RVFWS (CMR and echo) while the remaining imaging parameters did not explain the shortened walking distance. Resting measures of RV function thus appear to exert a limited impact on functional capacity in patients with PHpre-cap.

Functional capacity is only to a small extent determined by right ventricular function in

Table 1. Baseline characteristics for demographic, clinical, right heart catheterization and imaging parameters.

Baseline characteristics		
Number of patients (n)	49	
Age (years)	61±16	
Gender (women, n)	34	
BSA (m^2)	1.9±0.2	
Ethiology of pulmonary hypertension		
Pulmonary arterial hypertension (n)	40	
Chronic thromboembolic hypertension (n)	9	
Laboratory parameters		
NT-proBNP (ng/L)	1514 [50-6527]	
Saturation peripheral (%)	93 ± 4	
Right heart catheterization		
mPAP (mmHg)	40±12	
PCW (mmHg)	8±5	
Functional capacity		
6-MWT (m)	311±137	
Right ventricular function parameters		**R^2**
TAPSE (mm)	19±6	0.11*
S´$_{echo}$ (cm/s)	11.5±4	0.08
RVFAC$_{echo}$ (%)	30±13	0.08
RVFWS$_{echo}$ (%)	-15.0±4.3	0.17**
RVEF$_{CMR}$ (%)	39±13	0.13*
RVAVPD (mm)	13±4	0.23***
S'$_{CMR}$ (cm/s)	9±3	0.12*
RVFAC$_{CMR}$	33±10	0.07
RVFWS$_{CMR}$	-19.9±6.7	0.15**

All data are expressed as mean±SD or median [range]. mPAP (mean pulmonary pressure), PCW (pulmonary capillary wedge pressure), CMR (cardiac magnetic resonance), RVEF (RV ejection fraction derived from CMR), RVlatAVPD (RV lateral atrio-ventricular plane displacement), S´ (S´-wave velocity/max emptying velocity), RVFAC (RV fractional area change), RVFWS (RV free wall strain), TAPSE (tricuspid annular plane systolic excursion). R^2: Coefficients of determination to 6MWT; p-value * <.05, ** <.01, *** <0.001.

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Background:
Right ventricular (RV) failure is a major determinant of outcome in precapillary pulmonary hypertension (PH$_{pre-cap}$) and decreased exercise capacity is a marker of increased risk for death or lung transplantation. RV function can be assessed non-invasively by cardiac magnetic resonance imaging (CMR) or echocardiography (echo). New CMR measures of RV function, beyond RV ejection fraction (RVEF$_{CMR}$), have emerged but it is unknown if these are associated with functional capacity, i.e. 6-minutes walking test (6MWT). The aim was to evaluate if RV functional measures by CMR and their echocardiographic equivalents are determinants of 6MWT in PH$_{pre-cap}$ patients.
Methods:
Forty-nine patients (57% women, mean age 61±16 years) with PH$_{pre-cap}$ (pulmonary arterial hypertension (n=40) and chronic thromboembolic PH (n=9)) underwent prospective assessment of RV function at rest with CMR and echo. From CMR, RVEF$_{CMR}$, RV atrio-ventricular plane displacement (RVAVPD), maximum emptying velocity (S'$_{CMR}$), RV fractional area change (RVFAC$_{CMR}$) and RV free wall longitudinal strain (RVFWS$_{CMR}$) were measured. Equivalently echocardiographic measures of TAPSE, S´$_{echo}$, RVFAC$_{echo}$ and RVFWS$_{echo}$. were assessed. 6MWT was performed in conjunction with the exams. Coefficients of determination (R^2) was computed.
Results
Mean distance walked during 6MWT was 311±137 m (normal values 400-700m). RV functional impairment was shown for RVEF$_{CMR}$, RVFWS$_{echo}$ and RVFAC$_{echo}$. 6MWT was determined of RV$_{lat}$AVPD, RVFWS$_{echo}$, RVFWS$_{CMR}$, RVEF$_{CMR}$, S'$_{CMR}$ and TAPSE by 11-23% (Table 1), while S'$_{echo}$ and RVFAC$_{CMR}$ and RVFAC$_{echo}$ did not determine functional capacity.
Conclusion
6MWT was markedly decreased in patient with PH$_{pre-cap}$. However, functional capacity was only determined by a minor part of RV$_{lat}$AVPD, TAPSE, RVEF$_{CMR}$ and RVFWS (CMR and echo) while the remaining imaging parameters did not explain the shortened walking distance. Resting measures of RV function thus appear to exert a limited impact on functional capacity in patients with PH$_{pre-cap}$.

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Formaterat: Radavstånd: dubbelt

Validation of real-time phase contrast MRI with online compressed sensing reconstruction in phantom and patients

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The author has chosen not to publidh the abstract.

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Constructing ECG-free whole-heart cines from real-time cardiac magnetic resonance images acquired during free-breathing exercise

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The author has chosen not to publidh the abstract.

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Vascular simulation successfully guided and predicted the outcome of a complex Fontan intervention

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The author has chosen not to publidh the abstract.

4864-A-2301   P-31

Myocardial perfusion reserve assessed by quantitative CMR is lower in cardiac transplant patients than in healthy controls and is related to exercise capacity

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The author has chosen not to publidh the abstract.

Right ventricular stroke work index (RVSWI) by echocardiography in patients with pulmonary arterial hypertension – the role in follow-up assessment

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Background: Right ventricular failure is the main cause of mortality in patients with pulmonary arterial hypertension (PAH). Right ventricular stroke work index (RVSWI) measured by right heart catheterization (RHC) has been proposed as determinant of outcome, yet with conflicting results. Recently, in treatment naïve PAH patients, four different echocardiographically-derived RVSWI (RVSWIECHO1-4) has been compared with RHC derived RVSWI (RVSWIRHC), demonstrating a moderate to strong correlation. The aim of this study is to evaluate how RVSWIECHO changes at follow-up assessment of PAH patients testing these four methods.

Methods: In this study, 54 consecutive PAH patients (65 ± 13 years, 36 women) were analysed at baseline and at their first follow-up visit (3-6 months). Echocardiography and RHC were performed within a median of 0 day [IQR 0-1 days]. After baseline, PAH specific treatment was initiated. At follow up visit, RVSWIRHC was calculated using the formula: (mean pulmonary arterial pressure (mPAP)-mean right atrial pressure (mRAP)) x stroke volume index (SVI)RHC.

Four methods for RVSWIECHO were evaluated: RVSWIECHO-1=Tricuspid regurgitant maximum pressure gradient (TRmaxPG) xSVIECHO, RVSWIECHO-2=(TRmaxPG-mRAPECHO)xSVIECHO, RVSWIECHO-3=TR mean gradient (TRmeanPG)xSVIECHO and RVSWIECHO-4=(TRmeanPG-mRAPECHO)xSVIECHO. Estimation of mRAPECHO was derived from inferior vena cava diameter.

Results: Systolic and mean pulmonary artery pressure values, obtained by RHC, decreased significantly at follow-up visit ($p < 0.001$). There was a significant increase of SVI ($p < 0.01$) and a significantly reduced pulmonary vascular resistance ($p < 0.01$). In this interim analysis, RVSWI calculated invasively and with echocardiography, increased from baseline to follow-up, however none significantly. All data are presented in table 1.

Conclusion: Despite substantial improvement in conventional hemodynamic parameters from baseline to follow-up, none of the RVSWI methods showed statistically difference in patients with PAH.

Keywords: pulmonary hypertension, echocardiography, right ventricular stroke work index

Table 1.

Parameters	Baseline value	Follow-up value	Difference Baseline vs follow up visit	P-value
SPAP (mmHg)	75±19	64±20	-11±15	<0.001
mPAP (mmHg)	46±11	39±11	-7±9	<0.001
PCW (mmHg)	7±3	8±4	0.9±3.4	0.05
RAP (mmHg)	7±5	6±4	-0,9±4.4	0.09
SVI (ml/m^2)	30±8	37±10	7.5±.8.1	<0.01
CI (L/m^2)	3±0.8	2.9±0.7	-0.1±0.5	0.09
PVR	10±5	6±3	-3.9±3.7	<0.001
RVSWI$_{ECHO1}$ (mmHg*mL*m-2)	1896±594	1967±728	70±23	0.230
RVSWI$_{ECHO2}$ (mmHg*mL*m-2)	1722±569	1765±706	42±633	0.328
RVSWI$_{ECHO3}$ (mmHg*mL*m-2)	1092±372	1193±482	101±399	0.051
RVSWI$_{ECHO4}$ (mmHg*mL*m-2)	918±353	991±469	73±425	0.130
RVSWI$_{RHC}$ (mmHg*mL*m-2)	1131±386	1208±467	77±327	0.06

Data are presented as mean±SD. SPAP (systolic pulmonary arterial pressure), mPAP (mean pulmonary arterial pressure), PCW (pulmonary capillary wedge pressure), RAP (right atrial pressure), SVI (stroke volume index), CI (cardiac index), PVR (pulmonary arterial resistance), RVWSI (right ventricular stroke work index).

New methods for calculation of Right Ventricular Stroke Work Index by echocardiography in treatment naïve patients with pulmonary arterial hypertension

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Background:

Pulmonary arterial hypertension (PAH) is a disease with many different etiologies contributing to an increased pressure in the pulmonary circulation. The diagnosis of PAH is established by right heart catheterization (RHC). PAH contributes to pressure overload of the right ventricle (RV) resulting in impairment of RV function. Currently, RV stroke work index (RVSWI) by RHC is considered to be a measure of RV workload.

The purpose of the study is to compare two echocardiografically-derived measurements of RVSWI to RVSWI obtained by RHC (RVSWIRHC).

Methods:

This study included 54 consecutive treatment naïve patients with PAH (65 ± 13 years, 36 women). Echocardiography (ECHO) and RHC were performed within a median of 1 day [IQR 0-1 days]. RVSWIRHC was calculated as: (mPAP-mean right atrial pressure (mRAP)) x stroke volume index (SVI). Echocardiographically, two different methods for calculation of mPAP were used in the RVSWI formula: 1. RVSWIPAT= (90 - (0.62 x pulmonary acceleration time (PAT) x stroke volume index (SVIEKO)), 2. RVSWICHEMLA= ((0.61 x (tricuspid regurgitant maximum pressure gradient (TRmaxPG) + mRAP) + 2) x SVIECHO).

Results:

Both the echocardiographic methods showed a statistically significant difference (p <0.001) in absolute values compared to RVSWIRHC (Table 1). RVSWICHEMLA showed a moderate correlation (r=0.657, p <0.001) and RVSWIPAT showed no correlation (r=0.239, p=n. s) with RVSWIRHC.

Conclusion:

Echocardiographically-derived RVSWI calculated by using Chemlas formula for mPAP, demonstrated the best correlation with invasive RVSWI. The clinical applicability of echocardiographically-derived RVSWI warrant further studies.

Table 1

Tabel 1

Echocardiographic parameters	
$TR_{max}PG$ (mmHg)	69 ± 17
$mPAP_{PAT}$ (mmHg)	45.3 ± 13.1
$mPAP_{CHEMLA}$ (mmHg)	50.4 ± 12.1
mRAP (mmHg)	8 [3-8]
SVI (ml/m^2)	28.4 ± 8.7
$RVSWI_{PAT}$	1239 ± 455
$RVSWI_{CHEMLA}$	1390 ± 420
Right heart catheterization	
mPAP (mmHg)	47 ± 12
SVI (ml/m^2)	29.4 ± 8.4
$RVSWI_{RHC}$ (mmHg x mL/m^2)	1132 ± 352

Data are presented as mean±SD. TRVmaxPG (tricuspid regurgitant maximum pressure gradient), mRAP (mean right atrial pressure), $mPAP_{PAT}$ (mean pulmonary artery pressure calculated as : mPAP = 0,62xpulmonary acceleration time (PAT), $mPAP_{CHEMLA}$ (mean pulmonary artery pressure calculated by Chemlas formula, mPAP=0,61 x (tricuspid regurgitant maximum pressure gradient (TRmaxPG) + mRAP), SVI (stroke volume index), mPAP (mean pulmonary arterial pressure), RVSWI (right ventricular stroke work index).

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Lung vessel information improves automatic lung lobe segmentation in CT scans

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Background

Lung lobe segmentation has proved important for assessment of extent and distribution of lung volumes and disease. In expiratory pulmonary computed tomography (CT) scans automatic segmentation often fail due to the collapsed lung. The aim of this study was to investigate if information from lung vessels can improve lung lobe segmentation with deep learning (DL) methods. The hypothesis is that the lung vessels average direction varies in the different lung lobes and that this information can help the segmentation algorithm distinguish between the lobes.

Methods

We used inspiratory and expiratory CT scans from 59 research persons (a local cohort from the Swedish SCAPIS study). Annotated lung lobes, manually segmented by an expert radiologist served as ground truth. 48 paired scans were used for training the algorithm and 11 were used for evaluation. A map of the lung vessels was created by letting an in-house semi-automatic algorithm follow the pulmonary vessels through the lung, creating a 3D map of the vessels for every CT scan. A DL model, including the CT volume and the lung vessel map was trained to predict lobular segmentation. Results were compared to a baseline model, trained without lung vessel map. Results from the two algorithms were compared by Dice score (volumetric overlap) and average surface distance (ASD).

Results

Results were calculated as average for all lung lobes. ASD was decreased by 9.5% for expiration and 7% for inspiration, compared to baseline model. Dice score for inspiration was 0.97 (baseline) and 0.96 (lung vessel map), for expiration both models gave Dice score 0.96.

Conclusions

Our study confirms that relatively simple freely available deep learning methods, applied in pulmonary CT scans for lung lobe segmentation, do not deviate strongly from manual segmentation. The ASD score results suggest that adding lung vessel information may further improve the segmentation.

Left atrial deformation discloses atrial amyloid infiltration among patients with increased myocardial thickness.

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Background: Transthyretin cardiac amyloidosis (ATTR-CA) is an infiltrative disease associated with high risk of heart function impairment, caused by amyloid deposits in ventricles and atriums. We aimed to evaluate left atrial deformation including left ventricular-atrial strain loop (LV-LA loop). The latter to test LA deformation and its dependence of LV deformation in ATTR CA and compare it to left ventricular hypertrophy (LVH).

Methods: Retrospective analysis was performed on available echocardiographic data obtained between 2004-2022. The study population was divided into three groups: healthy controls (no identified cardiovascular disease), ATTR-CA (verified ATTR-diagnosis with end diastolic interventricular septum (IVSd) thickness ≥14mm) and LVH (including IVSd ≥14mm and no CA diagnosis). Left heart strain measurements, including LV global longitudinal strain (LV GLS) and LA peak atrial longitudinal strain (LA PALS), were acquired from apical 4-chamber view during same single heart cycle. Strain coordinates throughout the heart cycle were plotted using MATHLAB, combining LV GLS and LA PALS to compute a LV-LA loop. A slope was determined using regression line to index relative function of the left ventricle to left atrium (GLS-PALS-slope).

Results: Study population included 90 patients (68±1 years, 56% male), each group containing 30 patients. The lowest PALS and PALS-GLS slope was detected in ATTR-CA (p-value <0.05 compared to LVH). No significant difference was verified in GLS between ATTR-CA and LVH (p-value 0.114). A ROC curve for ATTR-CA demonstrated similar results between PALS, GLS-PALS slope and slope indexed with LAVI, all higher than GLS.

Conclusions: LA deformation demonstrates a strong ability to differentiate ATTR-CA from LV hypertrophy. Adding LV strain in relation to LA deformation showcases the mechanical dissociation of LA to LV in ATTR-CA, potentially unmasking atrial infiltration. Its use in prognostic evaluation needs further studies.

Clinical characteristics and echocardiographic findings

	CONTROL	ATTR-CA	LVH	p-value*
Male n (%)	14 (46,7)	19 (63.3)	17 (56,7)	0.598
Age	62.0 ± 9.7	74.6 ± 8.9	66.8 ± 10.9	0.004
IVSd (mm)	9.5 ± 1.8	19.1 ± 3.1	16.7 ± 2.7	0.002
PWT (mm)	7.8 (2.3)	13.5 (3)	10.0(2.0)	<0.001
E/A	1.0 (0.5)	1.0 (1.0)	0.9 (0.4)	0.292
E/é	8.1 (3.4)	13.8 (5.2)	11.7 (5.4)	0.13
LAVI (cm^3/m^2)	27.1 ± 10.6	41.6 ± 11.7	43.9 ± 23.4	0.65
LVEF (%)	60 (3)	56 (10)	59 (12)	0.167
GLS (%)	-18.1 (4.9)	-14.6 (6.1)	-16.0 (6)	0.114
PALS (%)	27.2 ± 7.4	13.5 ± 8.0	20.3 ± 10.0	0.005
GLS-PALS Slope	1.28 ± 0.43	0.70 ± 0.40	1.00 ± 0.35	0.003
Slope/LAVI Index	0.051 (0.028)	0.016 (0.013)	0.025 (0.023)	0.010

Values are as mean ± SD, or median (interquartile range). *The P values refer to comparison between the subgroups ATTR-CA and LVH.

IVSd = interventricular septum end diastole; PWT = posterior wall thickness; LVEF = left ventricular ejection fraction; GLS = global longitudinal strain; PALS = peak atrium longitudinal strain; LAVI = left atrium volume index

ROC Curve
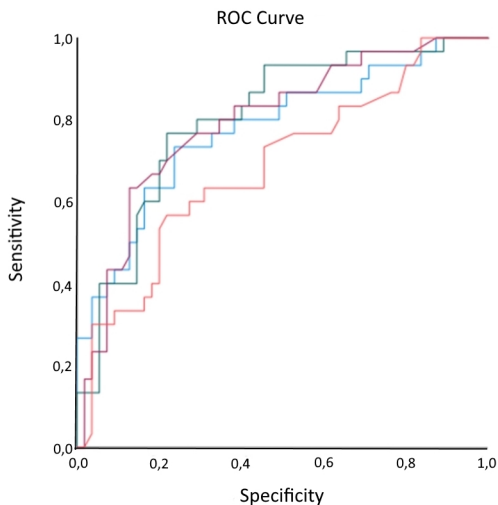
	AUC	95% IC
GLS	0.69	0.57 - 0-81
PALS	0.79	0.69 - 0.89
GLS-PALS Slope	0.78	0.67 - 0.88
Slope/LAVI Index	0.80	0.71 - 0.90

Echocardiographic assessment of left atrial reservoir strain the first year following heart transplantation

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Background

Left atrial (LA) reservoir strain measurement using echocardiography is promoted as a new tool for assessment of LA function correlating to left ventricular (LV) filling pressures. Disturbed diastolic function causes increased LA volume and impaired LA reservoir strain (LAr). Reduction in filling pressures results in a decreased (but rarely normalized) LA volume while LAr is reported as more sensitive in reflecting load-dependence. We aimed to study how LAr was affected between 3 and 12 months after heart transplantation (HTx). Furthermore, we aimed to explore the relationship between LAr and LA size and LV longitudinal strain (LVGLS) respectively.

Methods

Forty-four HTx-patients (33 males, mean age 47 ± 12 years) underwent transthoracic echocardiography (TTE) at 3 and 12 months post HTx. Strain was measured using Philips automated software (autostrain) with manual correction to ensure appropriate tracking. Data are expressed as mean $\pm$ SD. For correlations Pearson´s correlation coefficients was used.

Results

Between 3 and 12 months LAr improved from $-18.4\pm6.2\%$ to $-22.0\pm6.6\%$ ($p<0.001$), while LA volume or BSA-indexed LA (LAi) volume remained unaltered (p=n.s). Between the same time-points LVGLS increased from $-14.7\pm3.2\%$ to $-16.9\pm3.4\%$ ($p<0.001$). LAr was correlated to LA/LAi volume at 3 and 12 months (3 months: R= 0.30, $p<0.05$ and 0.41, $p<0.01$, 12 months: R= 0.30, $p<0.05$ and 0.32, $p<0.05$, respectively). At 12 months, LVGLS and LAr showed a moderate correlation (R= 0.51, $p<0.001$).

Conclusion

During the first year following HTx we observed a LAr enhancement unrelated to LA volume change. The increase in LAr was accompanied by an increase in LVGLS, and at 12 months moderate correlation between LAr and LVGLS was found while LA/LAi volume remained unaltered. This suggests that in HTx-patients LAr might poorly reflect LV filling pressures, but may rather be a mirror image of changes in LV function, here represented by LVGLS.

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The utility of left atrial reservoir strain measured by echocardiography for prediction of left ventricular filling pressure in heart transplant patients

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Background

Non-invasive assessment of left ventricular (LV) filling pressure is challenging. Although many parameters reflecting different parts of diastolic function exists, no single parameter can distinguish between grades of diastolic dysfunction. In heart transplant (HTx) patients, the echocardiographic evaluation is further complicated by altered physiology and geometry related to the surgical procedure. Echocardiographic left atrial reservoir strain (LAr) has been suggested as a novel parameter reflecting LV filling pressure. Consequently, we aimed to evaluate the potential role of this parameter post-HTx.

Methods

Forty-four HTx patients (33 males, mean age 47±12 years) underwent transthoracic echocardiography (TTE) in conjunction with right heart catheterization (RHC) 12 months after HTx. LAr was measured using Philips automated software (autostrain) with small manual correction to ensure appropriate tracking. Data are expressed as mean ± SD. The correlation between TTE and RHC was explored using Pearson´s correlation coefficients.

Results

LAr was -22.2±6.7%, left atrial (LA) volume was 75±23 ml and LA volume indexed to BSA was 35±14 ml. Invasively measured pulmonary artery wedge pressure (PAWP) was 7.4±3.4 mmHg and pulmonary vascular resistance (PVR) was 1.2±0.5 WU. The absolute value of LA reservoir strain was to some extent reflected by the LA volume (R= 0.30 for LA volume and R=0.32 for indexed LA volume, p<0.05 for both). No significant correlation between LAr and PAWP or PVR was found.

Conclusion

Our results suggests that LAr is a poor marker for prediction of LV filling pressure 12 months post-HTx. This could hypothetically be related to LA scarring together with changes in LA geometry/volume following transplant surgery. The relation between LA volume and LAr remains of interest since strain measures the degree of shortening compared to origin. Further validation to explore if indexed strain-values in relation to volume or length might add diagnostic value in clinical assessment.

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Indexed right ventricular strain - a novel echocardiographic measure showing correlation to invasively measured stroke volume in heart transplant patients

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Background

Evaluation of right ventricular (RV) function using transthoracic echocardiography is challenging due to multiple factors including the shape and localization of the RV. Speckle tracking derived strain has shown promising results for measuring longitudinal contractility, but does not take ventricular size into consideration. Amongst 2-dimensional (2D) measurements, the only one that incorporates radial motion is fractional area change (FAC) as it measures the percentage change in RV area. We aimed to investigate how RV function parameters correlate to stroke volume (SV) measured by right heart catheterization (RHC) and to explore if strain indexed to RV length and delta-FAC could have additional value.

Methods

Fifty heart transplant (HTx) patients (39 males, mean age 50 ± 12 years) were examined with TTE in conjunction with RHC 12 months after HTx. Indexed strain-values of the RV global longitudinal strain (GLS) and RV lateral wall strain (RVFree) were calculated by multiplying the RV length measured from the apical 4-chamber view with the obtained strain value. Delta-FAC was defined as the difference in area between diastole and systole. Pearson´s correlation coefficient was used to evaluate the relationship between echocardiographic RV function measures and SV obtained from RHC (SVRHC).

Results

We found no correlation between conventional echocardiographic measures of RV function (i.e. TAPSE, S´, FAC, RVGLS and RVFree) and SVRHC. However, a moderate correlation between both indexed RVGLS, RVFree and delta-FAC to SVRHC was detected (R=0.31, 0.36 and 0.35 respectively, $p<0.05$).

Conclusion

Conventional echocardiographic measurements of RV function, including strain, failed to mirror SVRHC. Our results indicate that RV length should be taken into consideration when using longitudinal echocardiographic measures of contractility, especially if the purpose is to reflect SV and hence to evaluate cardiac output. This assertion is supported by the finding that delta-FAC, which interrogates change in radial length, also showed a correlation to SVRHC.

Left Atrial Dissection a rare complication of mitral valve replacement

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Left atrial dissection is a very rare complication that occurs during cardiac surgery and has a high mortality rate. Mitral valve replacement is the most common suggested cause of left atrial dissection, and it occurs in less than 1% of all mitral valve replacements. There are few case reports and not a lot of data in the literature. The mechanism of atrial dissection is usually unknown, where our experience might bring important information regarding etiology and complications.