

Heart failure with a preserved ejection fraction additive value of an exercise stress echocardiography

Erwan Donal^{1,2,3*†}, Christophe Thebault^{1,2,3†}, Lars H. Lund⁴, Gaëlle Kervio^{2,3},
Amelie Reynaud^{2,3}, Tabasomne Simon⁵, Elodie Drouet⁵, Emilie Nonotte⁵,
Cecilia Linde⁴, and Jean-Claude Daubert^{1,2,3}

¹Department of Cardiology, Pontchaillou University Hospital, 2 Rue Henri Le Guilloux, 35033 Rennes, France; ²INSERM U642, Rennes, France; ³INSERM 804, Rennes, France;
⁴Department of Cardiology, Karolinska University Hospital, Stockholm, Sweden; and ⁵URC Paris Est, French Cardiology Society, Paris, France

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Background

Heart failure (HF) with a preserved (P) left ventricular (LV) ejection fraction (EF) is common, though its diagnosis and physiopathology remains unclear. We sought to analyse the myocardial characteristics at rest and during a sub-maximal exercise test in patients with HFPEF.

Methods and results

Standardized sub-maximal exercise stress echocardiography was performed in (i) 21 patients from the Karolinska Rennes Prospective Study of Heart Failure with Preserved Left Ventricular Ejection Fraction HFPEF registry, whose LVEF was $\geq 45\%$ and (ii) 15 control patients free of any manifestations of HF. During a sub-maximal exercise test, LV systolic function measured as a global four-chamber longitudinal strain was $-17 \pm 5\%$ in patients with HFPEF vs. $-22 \pm 4\%$ in controls ($P < 0.001$), LV longitudinal diastolic relaxation, expressed as e' (septal and lateral walls averaged) was 9 ± 2 cm/s in patients vs. 15 ± 4 cm/s in controls ($P < 0.001$), and RV longitudinal systolic function, expressed as RV s' , was 14 ± 3 cm/s in patients vs. 18 ± 1 cm/s in controls ($P = 0.03$). LV afterload (arterial elastance) was 2.7 ± 1 mmHg/mL and was correlated with a decrease in LV longitudinal strain ($R = 0.51$, $P < 0.01$) during exercise.

Conclusion

The assessment of longitudinal systolic and diastolic LV and RV functions is valuable during a sub-maximal exercise stress echocardiography to confirm the heart dysfunction related to the HFPEF symptoms. It might be used as a diagnostic test for difficult clinical situations.

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Keywords

Heart Failure • Ventricular Function • Exercise Echocardiography • Stress Echocardiography • Longitudinal Myocardial Function • Myocardial Strain

Introduction

Left ventricular (LV) ejection fraction (EF) is $>45\%$ over half of the patients presenting with manifestations of heart failure (HF).^{1–5} The diagnosis and prognosis of HF with a preserved (P) EF and factors contributing to its expression and pathophysiology are controversial and challenging, and its treatment is non-standardized.^{1–4}

Recent definitions of HF with a PEF usually include three criteria: (i) an LVEF $>45\%$, (ii) an LV end-systolic volume <97 mL/m², and (iii) a E/e' ratio of >15 .⁵

A sub-maximal exercise echocardiography (SEE) has been used in some studies to detect abnormalities of systolic and diastolic functions not apparent at rest.^{4,6,7}

The Karolinska Rennes Prospective Study of Heart Failure with Preserved Left Ventricular Ejection Fraction (KaRen) is a registry,

* Corresponding author. Tel: +33 2 99 28 25 25; Fax: +33 2 99 28 25 10, Email: erwan.donal@chu-rennes.fr

† The contribution of the first two authors is identical.

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which studies the clinical, laboratory, and echocardiographic characteristics and 18-month outcomes of patients presenting with HFPEF defined as an LVEF $\geq 45\%$ and prospectively included in France and Sweden.⁸ The present sub-study to KaRen registry sought to demonstrate the value of indices of heart longitudinal function (tissue Doppler and longitudinal strain) at rest and during a sub-maximal exercise test to explain symptoms of HF in patients without any obvious abnormality in heart function as compared with controls of the same age.

Patient population and methods

The design of the KaRen registry has been described previously.⁸ Prospectively, it includes consecutive patients presenting with (i) manifestations of acute HF according to the Framingham study criteria,⁹ (ii) a blood N-terminal pro-brain natriuretic peptide concentration >300 pg/mL, and (iii) an LVEF $\geq 45\%$ measured echocardiographically within first 72 h after admission to the hospital. The protocol of the KaRen registry was approved by the human research committees of both enrolling institutions, and all patients, including the controls, and gave a written informed consent to participate in the registry and its sub-studies.

Patients were recruited at our University Hospital for this sub-study. Patients were proposed to perform a supine exercise only after insuring the stability of their haemodynamic status (no functional or clinical sign of acute HF decompensation), the absence of neurological or orthopaedic limitations. No change in any treatment was done for the test. Beta-blockers treatment was not modified. This sub-study included ambulatory SEE and serological testing 4–8 weeks after stabilization. To facilitate the comparison with the control group, only patients in sinus rhythm and <85 years of age were considered for the study. The controls were recruited prospectively during the same time period and were patients who underwent evaluations for atypical chest pain without the evidence of myocardial ischaemia or HF. These controls were completely free of any history of HF or any significant coronary artery or valvular heart diseases and were not taking any cardiovascular medication.

Sub-maximal exercise test

Following clinical examination, arterial blood pressure measurement (Dinamap Procure Auscultatory 100), 12-lead electrocardiogram, and resting transthoracic echocardiography (Vivid 7, General Electric Healthcare, Horten, Norway), the patients underwent a standard supine exercise echocardiography on a tilting table with an electromagnetic cycle ergometer (Ergometrics). Exercise testing was started at an initial workload of 30 W, the workload being increased by increments of 20 W every 2 min. The pedaling rate was 60 rpm, the electrocardiogram was recorded continuously, and blood pressure was measured every 2 min both on exercise and during recovery from exercise. Exercise testing was interrupted promptly in the case of typical chest pain, limiting breathlessness, dizziness, muscular exhaustion, severe hypertension (systolic blood pressure of ≥ 250 mmHg), or significant ventricular arrhythmia. Blood pressure, ECG, and echocardiographic images were acquired at rest and for a heart rate (HR, 100–120/min) and at least five consecutive beats were recorded. The test

should have been considered abnormal if the patient presented one or more of the following criteria: angina, evidence of shortness of breath at low workload level (<50 W), dizziness, syncope or near-syncope, ≥ 2 mm ST segment depression in comparison to baseline levels, rise in systolic blood during exercise <20 mmHg, or a fall in blood pressure and complex ventricular arrhythmias. The exercise duration was planned to be (8–10) min for every patient.

Two-dimensional and tissue Doppler echocardiography

All patients underwent detailed echocardiographic examinations at rest and during the just-mentioned exercise with a Vingmed Vivid™ 7 (GE Healthcare, Horten, Norway). Symptom-limited exercise testing was performed on a semi-recumbent, tilting bicycle ergometer (Ergoline GmbH, Bitz, Germany) to a maximum HR of 120 bpm (i.e. a sub-maximal exercise test to maximize the frame rate). LV end-systolic and end-diastolic volume and LVEF were measured by the modified biplane Simpson's method from the apical four- and two-chamber views.¹⁰ The LV mass was calculated by Devereux's formula. Left atrial volume was calculated by the biplane area-length method from the apical four- and two-chamber views and indexed to the body surface area.¹⁰ The early filling (E) and atrial (A) peak velocities, and deceleration time of early filling and isovolumic relaxation time were measured from transmitral flow. All measurements were averaged over three beats.

Peak mitral annular myocardial velocity of the LV septal and lateral walls was recorded with a real-time pulse-wave tissue Doppler method, allowing the measures of the mean peak systolic (s'), early diastolic (e'), and late diastolic (a') velocities.^{11,12} The LV filling pressure was calculated as the ratio of early mitral diastolic inflow velocity to early diastolic mitral annular velocity (E/e').¹³ Peak annular right ventricular (RV) free-wall velocities (RV s' and RV e' for, respectively, peak systolic and early diastolic velocities) were measured by the same method. Tricuspid annular peak systolic excursion was calculated, using an M-mode echocardiography.¹⁴ The peak systolic pulmonary arterial pressure (PAP) was estimated using the Bernoulli formula according to the tricuspid maximal jet velocity.

Speckle tracking

LV longitudinal, radial and circumferential strains were assessed using the speckle tracking method.¹⁵ The apical four-, two-, and three-chamber images and short axis at the papillary muscles level images were analysed off line by tracing the endocardium in end-diastole, and the thickness of the region of interest was adjusted to include the entire myocardium. The software automatically tracks the myocardial deformation on the subsequent frame and the results are displayed graphically (Figure 1).

Derived parameters

Stroke volume was calculated by the aortic pulse-wave Doppler method, whereby the velocity time integral of the aortic annular flow was obtained by tracing the pulse Doppler profile and multiplied by the area of the aortic annulus.¹⁶

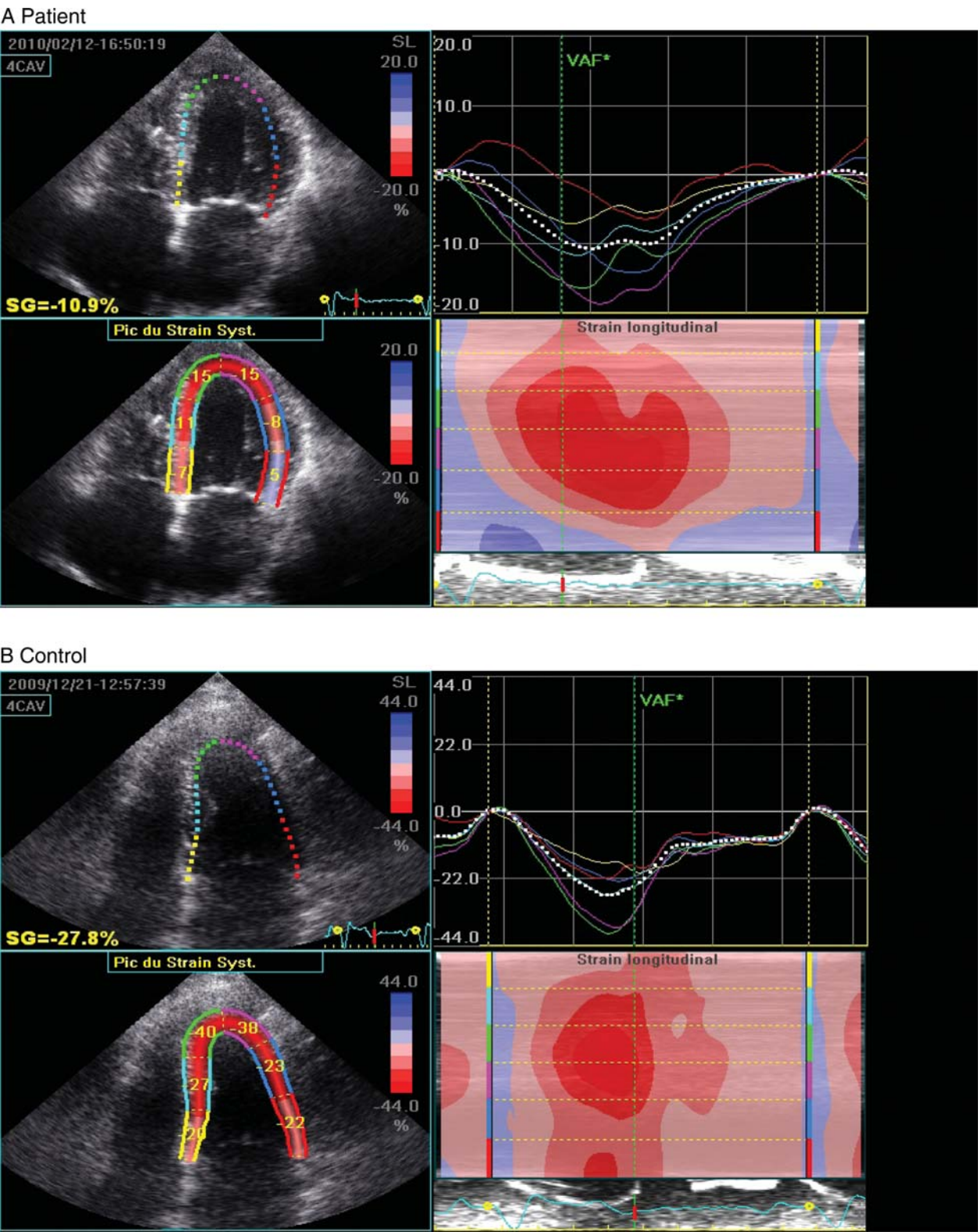


Figure 1 Two-dimensional strain in an apical four-chamber view in a patient with heart failure and preserved ejection fraction (A) and in a control patient (B).

Arterial elastance (A_e), expressed in mmHg/mL, was calculated to be $0.9 \times \text{systolic blood pressure}/\text{stroke volume}$, and end-systolic elastance (E_{es}), expressed in mmHg/mL, as $0.9 \times \text{systolic blood pressure}/\text{LV end-systolic volume}$.^{17,18} The 'meridional' wall stress, in dynes/cm², was calculated to be $0.334 \times (0.9 \times \text{systolic blood pressure}) \times \text{LV end-systolic diameter}/\text{systolic posterior wall thickness}$ [$1 + (\text{systolic posterior wall thickness}/\text{LV end-systolic diameter})$].^{4,19}

- Doppler tissue imaging (DTI) systolic longitudinal function reserve index = $\Delta s' \times [1 - (1/s' \text{ at rest})]$.⁴
- DTI early diastolic longitudinal function reserve index = $\Delta e' \times [1 - (1/e' \text{ at rest})]$.⁴

Longitudinal systolic reserve by speckle tracking imaging (STI) was calculated, using the apical four-chamber global longitudinal strain (GLS 4 chamber), as STI systolic longitudinal reserve = $\Delta \text{GLS 4 chamber} \times [1 - (1/\text{GLS 4 chamber at rest})]$.²⁰

Statistical analysis

The data, expressed as means \pm standard deviation (SD), were analysed with parametric statistics, after mathematical confirmation of normal distribution with the Shapiro–Wilk test.

Table 1 Clinical, biological, and electrocardiographic characteristics parameters

	Patients (n = 21)	Controls (n = 15)	P
Age (years)	76 \pm 6	75 \pm 5	0.03
Men, n (%)	12 (57)	8 (53)	ns
Body mass index (kg/m ²)	29 \pm 6	30 \pm 4	
Medical history			
Hypertension	19/21 (90)	9/15 (60)	<0.05
Diabetes	5/21 (24)	2/15 (13)	ns
Atrial fibrillation	0/21 (0)	0/10 (0)	<0.01
Coronary artery disease	5/21 (23)	0/10 (0)	
Valvular disease	3/21 (14)	0/10 (0)	
Drug regimens			
ACE-i or AT-1 blocker	12 (57)		
Beta-adrenergic blocker	17 (81)		
Calcium-channel blocker	7 (33)		
Diuretic	20 (95)		
Antiplatelet agent	11 (52)		
Vitamin K antagonist	8 (30)		
Serum creatinine ($\mu\text{mol/l}$)	102 \pm 36		
N-terminal-proBNP (pg/ml)	1984 \pm 2159		
Resting systolic blood pressure (mmHg)	146 \pm 36	145 \pm 18	ns
Left bundle branch block	3 (14)		
QRS duration (ms)	100 \pm 12		

Values are means \pm SD or numbers (%) of observations.

Between-groups characteristics were compared by unpaired *t*-test. Within groups measurements made at rest vs. exercise were compared by paired *t*-test. Relationships among selected variables were examined with Pearson's product moment correlation. Statistical significance was set at $P < 0.05$ for all analyses.

Results

Baseline characteristics

The baseline characteristics of both study groups are listed in Table 1. No significant change in age and rest HR was observed between the controls and the HF patients. The patients were predominantly male, hypertensive, and overweight. The mean resting blood pressure was 146/74 ($\pm 36/8$) vs. 145/80 ($\pm 18/10$) in controls. The median NT-proBNP in the HF group, at the time of the stress echocardiography was 1984 pg/mL (± 2159 pg/mL) and was not measured in controls. Controls did not have a history of HF or breathlessness. Their ECG was normal.

The resting echocardiographic measurements are listed in Table 2. The left atrial volume index was higher in patients with HFPEF than that in controls ($P < 0.001$). LV end-diastolic volume index, wall thickness and LV myocardial mass index were higher than that in our HFPEF patients.

Haemodynamic changes

The haemodynamic measurements are shown in Table 3. Resting HR and systolic blood pressure were similar in patients with HFPEF and in controls, at the time of the examination. Exercise cycle length was 600 ± 77 ms in controls vs. 786 ± 152 ms ($P < 0.001$) in HF patients. The workload was 45–60 W in the HF

Table 2 Echocardiographic measurements at rest

	Patients (n = 21)	Controls (n = 15)	P
Diastolic interventricular septal thickness (mm)	11.8 \pm 2.5	11.3 \pm 1.7	ns
Diastolic posterior wall thickness (mm)	10.9 \pm 2.1	10.4 \pm 1.5	ns
Left ventricular			
End-diastolic diameter (mm)	51.5 \pm 7.1	45.6 \pm 4.1	0.004
Indexed LV mass (g/m ²)	133 \pm 37	96 \pm 27	0.001
End-diastolic volume index (mL/m ²)	61 \pm 20	46 \pm 9	0.006
Ejection fraction (%)	56 \pm 11	66 \pm 6	<0.05
Left atrial volume index (mL/m ²)	41 \pm 12	33 \pm 6	<0.001
E-wave (cm/s)	96 \pm 28	79 \pm 23	0.04
A-wave (cm/s)	73 \pm 25	78 \pm 17	ns
Diastolic time (ms)	229 \pm 94	208 \pm 59	ns
IVRT (isovolumic relaxation time) (ms)	95 \pm 22	88 \pm 20	ns
E/e'	13 \pm 6	8 \pm 2.5	0.0002

Table 3 Haemodynamic status and Doppler echocardiography

	Patients			Controls			
	Rest	Exercise	P*	Rest	Exercise	P*	P**
HR (bpm)	61 ± 16	85 ± 23	<0.001	64 ± 12	103 ± 15	<0.001	ns ^a ; <0.05 ^b
Systolic blood pressure (mmHg)	146 ± 35	187 ± 25	<0.001	145 ± 18	186 ± 19	<0.001	ns ^a ; ns ^b
Δsystolic blood pressure (mmHg)		47 ± 29			36 ± 18		ns; 0.08
Stroke volume (ml)	62 ± 19	66 ± 24	ns	53 ± 14	62 ± 16	0.03	ns ^a ; ns ^b
Δstroke volume (ml)		6 ± 18			4 ± 17		ns
Left ventricular ejection fraction (%)	56 ± 11	59 ± 14	ns	66 ± 6	69 ± 8	ns	0.001 ^a ; 0.01 ^b
Δleft ventricular ejection fraction (%)		2 ± 9			3 ± 10		ns
Velocity–time integral (cm)	23 ± 6	22 ± 6	ns	22 ± 3	23 ± 2	0.05	ns ^a ; ns ^b
Δvelocity–time integral (cm)		−1.1 ± 4			1 ± 6		ns
Cardiac output (l/min)	5.0 ± 1.4	6.6 ± 1.7	<0.001	5.2 ± 1.5	8.0 ± 1.7	<0.01	ns ^a ; 0.02 ^b
Δcardiac output (l/min)		1.79 ± 2.47			3.04 ± 1.92		0.05
e' (cm/s)	7 ± 2	9 ± 2	<0.001	10 ± 2	15 ± 4	0.001	0.01 ^a ; <0.001 ^b
Δe' (cm/s)		1.8 ± 2.2			4.8 ± 2.8		0.008
E/e'	13 ± 6	15 ± 6	0.05	8 ± 2	16 ± 6	0.001	<0.05 ^a ; ns ^b
ΔE/e'		1.1 ± 4.3			7.1 ± 8		0.007
s' (cm/s)	7 ± 1	8 ± 2	0.001	9 ± 2	12 ± 2	<0.01	<0.001 ^a ; <0.001 ^b
Δs' (cm/s)		0.9 ± 1.7			1.4 ± 1.1		ns; 0.18
Tricuspid regurgitant velocity (m/s)	2.8 ± 0.4	3.4 ± 0.6	ns	2.4 ± 0.1	3.2 ± 0.4	ns	ns ^a ; ns ^b

^aPatients vs. controls at rest.^bPatients vs. controls during exercise.

*Paired t-test; **unpaired t-test.

and control groups (sub-maximal exercise test to reach the pre-defined target HR <120). Stroke volume at rest and during exercise and its increase during exercise, were not statistically different between study groups (Table 3). During exercise, however, aortic outflow velocity time integral did not change significantly in patients with HFPEF, but increased significantly in controls ($P = 0.05$). Thus, the increase in cardiac output during exercise was higher in the control than that in the HF group ($P = 0.02$) because the HR increment and workload were higher in controls than that in HFPEF patients. There are several explanations to this: drugs such as beta-blockers and also the inability to raise HR. The increase in systolic BP was higher ($P = 0.08$) in HFPEF patients (47 ± 29 mmHg) vs. 36 ± 18 mmHg in controls. What really happens here is that the HFPEFs have a relatively fixed stroke volume due to filling pattern and hence cannot increase HR.

Longitudinal LV function: tissue Doppler and speckle tracking

Mitral annular velocities in systole (s') and early diastole (e') at rest and during exercise were significantly lower in patients with HF than that in controls at rest and during exercise (Table 3). The absolute difference in s' between rest and exercise was similar in both groups, while the change in e' during exercise was significantly greater in controls (Figure 2).

By STI, the four- and three-chamber GLSs at rest were significantly lower in the HF than that in the control group (Table 4), whereas during exercise, the four-chamber GLS increased more

in the control group (Figure 3). But, the longitudinal systolic reserve measured by DTI or STI (s' reserve index or 2DS systolic reserve) did not reach any statistical significance between both groups. The early longitudinal diastolic reserve (e' reserve index) was significantly higher in the control than that in the HF group (Table 4).

Radial and circumferential LV systolic function: 2-D strain

The global radial strain was similar in both groups at rest and during exercise (Table 4), whereas the circumferential strain was significantly higher under both conditions in the control than that in the HF group (Figure 4).

Longitudinal RV function

RV s' at rest was significantly higher in the control than that in the HF group, while RV e' at rest was similar in both groups (Table 5). The free-wall annular tricuspid velocities in systole (RV s') and early diastole (RV e') during exercise were significantly higher in the control than that in the HF group.

Derived parameters

The LV meridional wall stress was quite stable from rest to exercise in controls (569 ± 189 dynes/cm²). It was higher in HFPEF patients (835 ± 407 dynes/cm², $P = 0.015$) compared with controls at rest and it increased clearly during exercise (927 ± 485 dynes/cm², $P = 0.003$). E_{es} was significantly lower in the HF

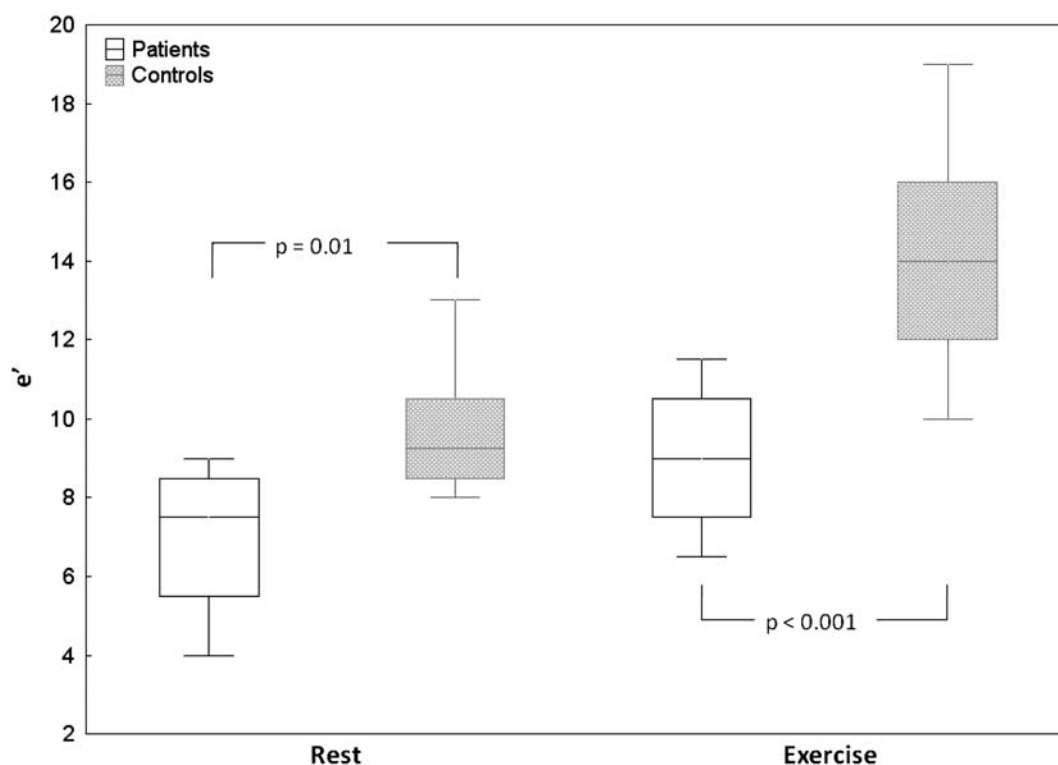


Figure 2 e' in patients with heart failure and preserved ejection fraction vs. controls.

Table 4 Two-dimensional strain and longitudinal reserve

	Patients			Controls			P^{**}
	Rest	Exercise	P^*	Rest	Exercise	P^*	
Global 4-chamber longitudinal strain (%)	-16 ± 5	-17 ± 5	ns	-20 ± 3	-23 ± 4	0.006	0.005 ^a ; 0.0005
Δ global 4-chamber longitudinal strain		-1.2 ± 4.0			-2.4 ± 2.1		ns
Global 2-chamber longitudinal strain (%)	-16 ± 4	-18 ± 5	ns	-19 ± 3	-22 ± 4	0.05	0.01 ^a ; <0.01 ^b
Δ global 2-chamber longitudinal strain		-2.8 ± 5.0			-3.2 ± 3.3		ns
Global 3-chamber longitudinal strain (%)	-16 ± 4.5	-17 ± 5	ns	-20 ± 3	-21 ± 3	ns	<0.01 ^a ; 0.01 ^b
Δ global 3-chamber longitudinal strain		-1.5 ± 3.6			0.5 ± 2.4		ns
Global radial strain (%)	36 ± 21	33 ± 19	ns	37 ± 16	38 ± 23	ns	ns ^a ; ns ^b
Global circumferential strain (%)	-14 ± 4	-15 ± 4	ns	-19 ± 2	-21 ± 3	ns	<0.01 ^a ; <0.001 ^b
Δ global circumferential strain		0.4 ± 4.5			-1.6 ± 3.3		ns
DTI systolic reserve index		0.79 ± 1.51			0.79 ± 2.11		ns
DTI early diastolic reserve		1.65 ± 1.76			3.34 ± 4.32		0.04
2-D strain systolic reserve		-1.36 ± 4.31			-2.64 ± 2.22		ns

^aPatients vs. controls at rest.

^bPatients vs. controls during exercise.

*Paired t-test; **unpaired t-test.

than the control group at rest and during exercise. However the delta of change during exercise was similar in both groups Table 6.

E_a/E_{es} was correlated with LV-GLS at rest [$R = 0.55$ ($R^2 = 0.30$), $P = 0.002$] and during exercise [$R = 0.49$ ($R^2 = 0.24$), $P = 0.006$].

E_a/E_{es} was also correlated with e' at rest ($R = -0.48$ ($R^2 = 0.23$), $P = 0.007$) and during exercise [$R = -0.46$ ($R^2 = 0.22$), $P = 0.01$]. The LV meridional wall stress was correlated with the LV longitudinal strain [$R = 0.33$ ($R^2 = 0.11$), $P = 0.04$] and e'

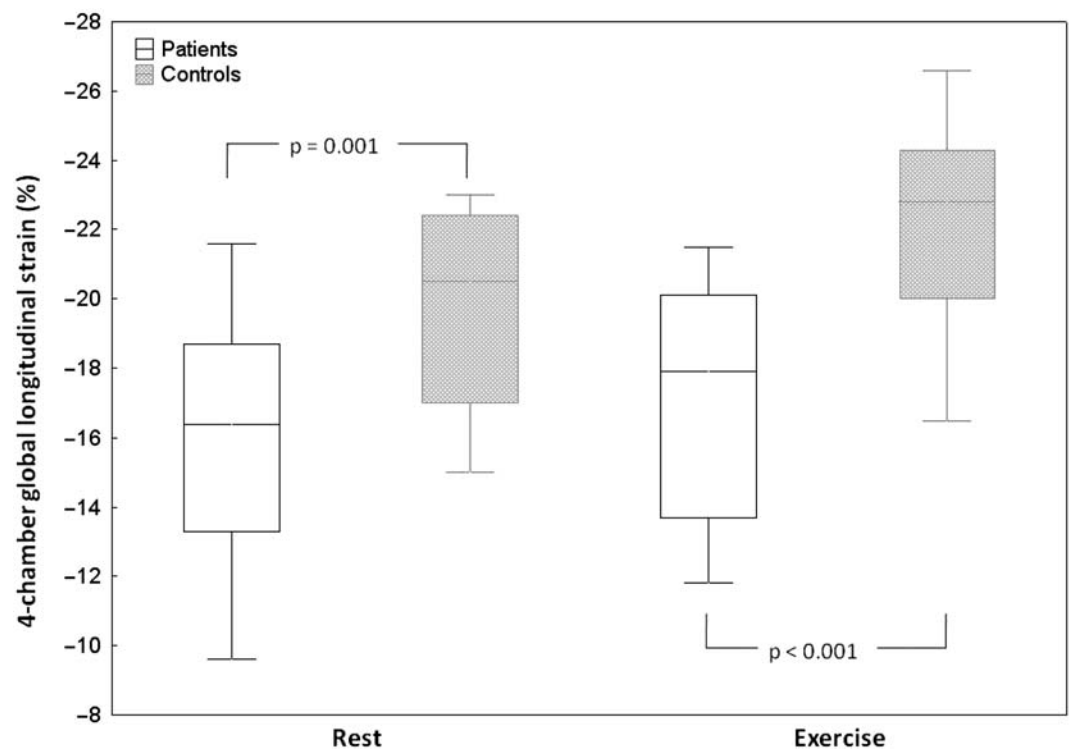


Figure 3 Four-chamber global longitudinal strain in patients with heart failure and preserved ejection fraction and in controls.

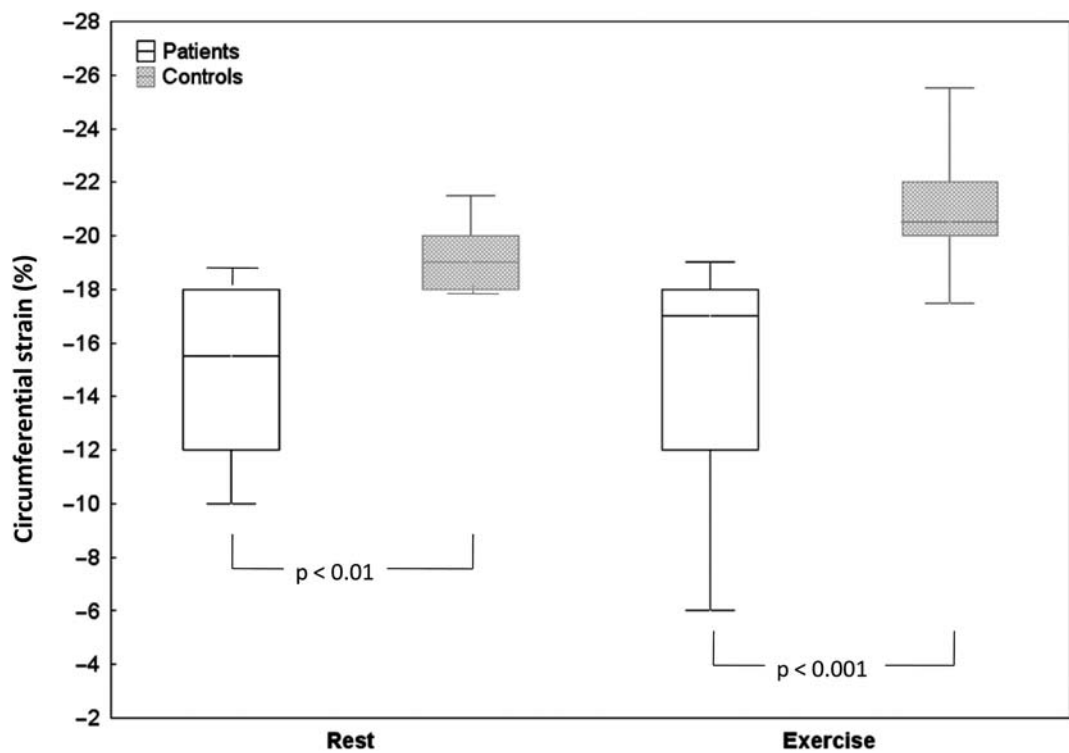


Figure 4 Circumferential strain in patients with heart failure and preserved ejection fraction and in controls.

Table 5 RV function parameters

	Patients			Controls			
	Rest	Exercise	P*	Rest	Exercise	P*	P**
RV s' (cm/s)	12 ± 3	14 ± 3	<0.001	14 ± 3	18 ± 1	ns	0.02 ^a ; 0.03 ^b
RV e' (cm/s)	9 ± 3	11 ± 5	<0.01	12 ± 4	20 ± 4	ns	0.01 ^a ; 0.001 ^b
Tricuspid annular peak systolic excursion (mm)	20 ± 5	22 ± 6	ns	24 ± 4	25 ± 4	ns	ns ^a ; ns ^b

^aPatients vs. controls at rest.^bPatients vs. controls during exercise.

*Paired t-test; **unpaired t-test.

Table 6 Derived parameters

	Patients			Controls			P**
	Rest	Exercise	P*	Rest	Exercise	P*	
Arterial elastance (mmHg/mL)	2.2 ± 0.9	2.7 ± 1.0	ns	2.6 ± 0.7	2.8 ± 0.7	ns	ns ^a ; ns ^b
Δarterial elastance (mmHg/mL)		0.5 ± 0.7			0.2 ± 0.7		ns
Meridional wall stress (dynes/cm ²)	835 ± 407	927 ± 485	0.04	569 ± 189	534 ± 176	ns	0.01 ^a ; <0.003 ^b
End-systolic elastance (mmHg/mL)	3.0 ± 2.2	4.2 ± 2.4	0.003	5.3 ± 1.8	7.1 ± 3.0	0.002	0.002 ^a ; <0.003 ^b
Δend-systolic elastance (mmHg/mL)		0.5 ± 3.5			1.7 ± 1.9		ns
Arterial elastance/end-systolic elastance	0.8 ± 0.3	0.8 ± 0.5	ns	0.5 ± 0.1	0.4 ± 0.2	ns	0.001 ^a ; 0.006 ^b

^aPatients vs. controls at rest.^bPatients vs. controls during exercise.

*Paired t-test; **unpaired t-test.

during exercise [$R = -0.33$ ($R^2 = 0.11$), $P = 0.04$]. E_a was correlated with LV-GLS but only during exercise with [$R^2 = 0.26$ ($R = 0.51$, $P < 0.01$)].

Discussion

Compared with controls, patients presenting with HFPEF suffer from systolic and diastolic dysfunction already at rest and which is accentuated during a sub-maximal exercise test. The assessment of longitudinal systolic and diastolic LV and RV functions might be used as a diagnostic test for difficult clinical situations, during a sub-maximal exercise stress echocardiography, to make sure that a heart dysfunction could explain HF symptoms.

Systolic function

The HFPEF group presented with a reduced longitudinal function already at rest, ascertained by measurements of myocardial velocity as well as by deformation imaging. This is not necessarily specific to the HFPEF. But, interestingly, exercise amplified the abnormality of LV systolic longitudinal function. We observed, in HFPEF, a depressed LV longitudinal function without reserve in HFPEF, as opposed to the normal pattern of increase in LV longitudinal strain seen in the controls. Our observations are concordant with recently published studies showing the importance of afterload on the depression of LV systolic longitudinal function.^{4,21–24} Like Tan et al.,⁴ the rest left ventricular end-diastolic

diameter and stroke volume were surprisingly higher in HFPEF patients, but the stroke volume did not increase as in control during the exercise. It has been demonstrated that longitudinal shortening is mainly dependent on the sub-endocardial fibres.²⁵ Also, this component of LV function is affected by a reduced sub-endocardial flow reserve and fibrosis,²⁶ though is also sensitive to afterload.¹⁹ In an animal model, the increase in afterload alters significantly the longitudinal component of LV systolic function.¹⁹ GLS and meridional wall stress were correlated. Furthermore, an increased LV rotational function has been described, which compensates for the decrease in longitudinal shortening in patients presenting with HFPEF.²⁷ It could be interesting to study the rotation during the exercise but we were not able to accurately measure this torsion, especially during exercise. We were however able to observe a significant alteration in the circumferential component of LV deformation (Figure 4), and similar radial strains at rest and during exercise in both groups. This might be partly explained by a compensatory effect, as occurs in hypertrophic disorders.²⁸ Of note, the absence of increase in radial deformation in the radial direction during exercise might be explained by technical limitations and warrants further studies, perhaps with 3-D speckle tracking.²⁹

Diastolic function

HFPEF was initially called diastolic HF.³⁰ However, there is growing evidence in favour of impairment of systolic as well diastolic

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