Abstract P6473 – Table 1. Comparison of SWS in different groups

SWE Parameter	Patients with CVEs (n=10)	Patients without CVEs (n=44)	p-value	Patients on Statin therapy <5yrs (n=16)	Patients on Statin therapy ≥5yrs (n=31)	p-value
SWSL median, m/s	2.26 (1.81–3.07)	3.51 (2.39–4.52)	0.04	2.50 (1.84–3.91)	3.52 (2.57–4.92)	0.07
SWST median, m/s	2.66 (2.06–3.41)	2.24 (1.94–2.91)	0.25	2.56 (2.03–3.35)	2.19 (1.74–2.81)	0.25
LTR Median	0.89 (0.75–1.28)	1.29 (1.05–2.17)	0.01	1.09 (0.81–1.30)	1.60 (1.05–2.19)	0.01
SWSL Mean, m/s	3.05 (2.43–4.86)	4.12 (3.13–5.16)	0.15	3.12 (2.81–4.92)	4.35 (3.43–5.54)	0.07
SWST Mean, m/s	3.24 (2.72–4.01)	2.87 (2.56–3.89)	0.48	3.07 (2.63–4.23)	2.87 (2.46–3.85)	0.39
LTR Mean	0.97 (0.79–1.32)	1.32 (0.99–2.00)	0.13	1.08 (0.70–1.46)	1.42 (1.08–2.08)	0.03

Data are expressed as the median (25th–75th percentile). SWSL: Longitudinal SWS; SWST: Transverse SWS; LTR: Ratio of Longitudinal and transverse SWS.

63% CAD history, 87% on statin therapy) were recruited. During 86±33 weeks follow up, 10 unique CVEs occurred (2 deaths, 3 MI, 3 UA and 2 TIA/CVA). The median SWSL and LTR were significantly lower in patients with CVEs vs. those without CVEs. Both mean and median LTR were significantly lower in patients on <5yrs statin therapy (table).

Conclusion: A decrease in the ratio of longitudinal to transverse SWS (LTR) was associated with CVEs suggesting less stiff (softer) plaques as defined by SWS, noninvasively characterize plaque vulnerability. Longer term statin therapy was associated with higher SWS (LTR), consistent with increased plaque stiffness. Thus, SWE may be useful to assess plaque vulnerability and efficacy of statin therapy.

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Clustering hypertension and overweight are synergistically associated with much larger left atrial volume. data from 3762 healthy individuals

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Background: Enlarged left atrial volume index (LAVI) is an important predictor of cardiovascular events. However, to what extent LAVI is progressively increasing with ageing and what kind of predisposing factors contribute to the enlarged LAVI have not been elucidated based on a large sample study.

Methods: A total of 3762 male and female individuals, aged 50 years or older without a history of stroke, coronary artery disease, atrial fibrillation, mitral stenosis or mitral regurgitation, who underwent a comprehensive physical check-up were enrolled. LAVI was estimated by the ellipsoid method using echocardiography. Participants were divided into several groups according to age, sex and predisposing factors (see table).

Results: Estimated means (95% confidence intervals) of LAVI are shown in the table. The higher LAVI was observed with an increment of age category independently of predisposing factors. Hypertension and obesity were associated with the higher LAVI and clustering predisposing factors were synergistically associated with much higher LAVI especially in older individuals.

table. Left atrium volume index (ml/m^2) in subjects stratified by age , sex and predisposing factors

age group	no factors		hypertension		overweight		diabetes		2 or 3 factors	
	n	mean (95%CI)	n	mean (95%CI)	n	mean (95%CI)	n	mean (95%CI)	n	mean (95%CI)
male subjects										
50-59	342	16.7 (16.2-17.1)	151	17.0 (16.3-17.7)	173	18.6 (17.9-19.4) †	37	16.6 (15.0-18.2)	251	19.8 (19.1-20.5) †
60-69	328	17.3 (16.7-17.8)	194	19.2 (18.3-20.0) †	129	20.8 (19.8-21.7) †	59	18.0 (16.3-19.7)	328	20.9 (20.3-21.5) †
70+	110	18.1 (17.1-19.1)	98	20.6 (19.2-22.0) †	44	21.5 (19.3-23.7) †	28	17.1 (15.2-18.9)	106	22.8 (21.7-23.9) †
female subjects										
50-59	256	16.0 (15.5-16.5)	59	16.8 (15.6-17.9)	66	18.0 (16.9-19.0) †	10	15.3 (11.8-18.9)	63	19.3 (18.2-20.4) †
60-69	289	16.4 (15.8-16.9)	138	19.4 (18.4-20.4) †	78	20.2 (19.0-21.3) †	23	18.6 (14.7-22.5)	126	21.6 (20.5-22.6) †
70+	102	19.3 (18.2-20.5)	78	19.4 (18.1-20.6)	30	19.7 (17.9-21.4)	4	17.0 (12.9-21.2)	62	24.0 (22.1-25.8) †

Data are expressed as means (95% confidence intervales)
hypertension: SBP≥140 or DBP≥90 or medication; overweight: BMI ≥25kg/m2; diabetes: FPG≥126mg/dl or PPG≥200mg/dl or HbA1c (NGSP)≥ 6.5% or medication.
†: <0.05 compared to the mean values in subjects with no factors.

Conclusion: This cross-sectional study indicated that LAVI is increasing with ageing independently of predisposing factors, however, clustering predisposing factors strongly contribute to the much larger LAVI with advance of age and this might lead to the future cardiovascular events through a process of developing atrial fibrillation.

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Global longitudinal strain as a prognostic marker in patients with normal left ventricular ejection fraction: a systematic review and meta-analysis

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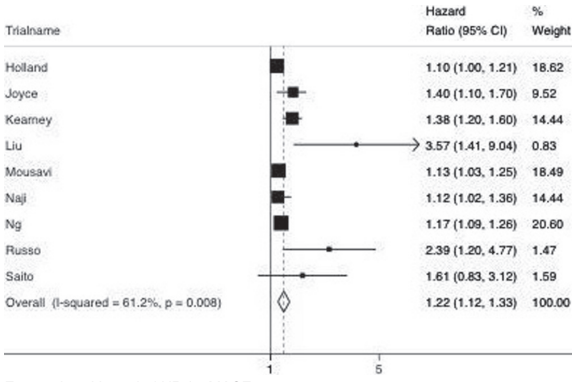
Background: Evidence suggests that global longitudinal strain (GLS) measured by speckle tracking echocardiography in patients with heart failure predicts risk of mortality and adverse cardiovascular outcomes. However, the prognostic significance of abnormal GLS in subjects with normal left ventricular ejection fraction (LVEF) without clinical heart failure remains unclear.

Purpose: To assess the association between abnormal GLS and clinical outcomes in subjects with normal LVEF.

Methods: EMBASE, MEDLINE, Cochrane Central Register of Controlled Trials (CENTRAL) and trial registries were searched systematically for studies investigating an association between GLS and clinical events in patients with normal LVEF (≥50%). The primary outcome was the composite of major adverse cardiovascular outcomes (MACE) (including all-cause mortality, cardiovascular mortality, vascular death, myocardial infarction, new heart failure, heart failure hospitalisation, target vessel revascularisation and/or ischemic stroke as defined by individual studies) or all-cause mortality. Two independent reviewers followed PRISMA protocol to extract the data and the review protocol was published in PROSPERO register (CRD42018086519).

Results: Nine studies involving 2767 patients (mean age 67±1 years, 54% male and BMI 28±5 kg/m^2) were included in the final quantitative analysis. Prevalence of cardiovascular risk factors were: hypertension (57%); diabetes (28%); smoking (25%); hypercholesterolemia (34%); and past history of coronary artery disease (30%). Thirty percent of the population used angiotensin-converting-enzyme inhibitors or angiotensin-II-receptor blockers and 29% were on beta-blockers. Mean pooled cut-off across the studies for abnormal GLS was -16%. During median follow up of 3.4±2.8 years, abnormal GLS was associated with MACE (HR 1.22, 95% confidence interval (CI) 1.12- 1.33, I^2 61.2%) (Figure 1) and all-cause mortality (HR 1.33, 95% CI 1.07- 1.66, I^2 78.3%).



Forest plot with pooled HR for MACE

Conclusion: In patients with normal LVEF, abnormal GLS predicts the risk of all-cause mortality and MACE. Since strain imaging is an accessible and practical technique it may be useful in assessing early left ventricular systolic dysfunction in the context of normal LVEF. Further prospective controlled studies of GLS in predicting adverse outcomes among homogeneous population are required.

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Multi-layer and segmental longitudinal strain response to incremental cycle exercise

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Introduction: Limited research exists identifying the myocardial layer and segmental longitudinal strain response to incremental cycle exercise stress echocardiography (ESE).

Purpose: To define the normal multi-layer and segmental longitudinal strain (LS) response to incremental cycle exercise in individuals without evidence of cardiovascular disease.

Methods: 30 healthy participants performed a commonly used ESE protocol. Images were obtained at rest, peak exercise and recovery. Post hoc analyses of data was completed using EchoPac (GE Medical, Norway). LS was calculated for the endocardium, mid-myocardium and epicardium, globally and in the base, mid and apical segments at rest, peak exercise and recovery. Myocardial layer (LSG) and base-apex strain (BASG) gradients were calculated. Analysis of variance and post-hoc Tukey's test were used to determine significant differences ($p<0.05$).

Results: LS increased significantly in all myocardial segments and layers from rest to peak exercise, rest to recovery and significantly decreased from peak exercise to recovery. GLS increased significantly in the endocardium and mid-myocardium from the base to the apex but not in the epicardium. There is a significant increase in LSG from basal-mid and mid-apical segments during rest, exercise and recovery. There is a significant increase in LSG between rest-exercise and rest-recovery in the apical segment only. BASG decreases significantly at rest, during exercise and recovery in all layers, there are no significant differences seen during rest, exercise or recovery in BASG within individual layers.