124 STROKE VOLUME DETERMINED FLOW RESERVE DOES NOT PREDICT THE TRUE SEVERITY OF LOW-FLOW LOW-GRADIENT AORTIC STENOSIS AND IS NOT A ROBUST MARKER OF CONTRACTILE RESERVE IN PATIENTS UNDERGOING LOW-DOSE DOBUTAMINE ECHOCARDIOGRAPHY

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Background During low-dose dobutamine stress echocardiography (LDDSE) in low-flow low-gradient aortic stenosis (LFLGAS), both the aortic stenosis (AS) severity and the presence of contractile reserve (CR) are conventionally assessed based on stroke volume flow reserve (SVFR), which is defined as stroke volume [SV] increase 20%. However frequent exaggerated chronotropic response to dobutamine with shortening left ventricular time result in SV drop. On the contrary, transvalvular flow rate (FR) (SV/ejection time) and left ventricular ejection fraction (LVEF) may increase. We aimed to assess the value of FR 200 ml/s (normal FR) and LVEF change in the identification of true severe AS (TSAS) and the assessment of CR respectively.

Methods Accordingly 74 consecutive patients (mean age 78 years) with LFLGAS referred for LDDSE for determination of AS severity and CR underwent retrospective assessment of SV, FR, LVEF and standard echocardiographic parameters of AS severity (Table 1). The outcome assessed was all-cause mortality censored for aortic valve intervention.

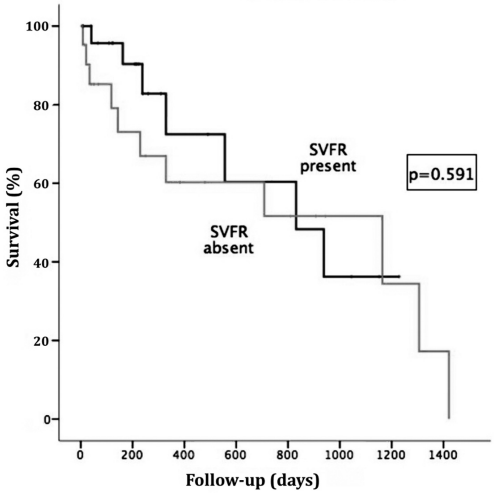
Results SVFR was present in 30 (40.5%) of the 74 patients whereas FR 200 ml/s was achieved in 60 (81.1%) (p<0.001). During the median follow-up of 316.5 days 28 (37.8%) deaths occurred. Amongst all standard echocardiographic predictors of AS severity at peak stress (aortic valve mean and peak gradient, peak velocity and area [AVA]) and clinical prognostic factors, AVA was an independent predictor of death (HR=0.1, 95%CI=0.02–0.7, p=0.03), and was therefore used to define TSAS (stress AVA 1.01cm^2). TSAS was present in 47 (63.5%) patients of whom SVFR correctly identified 17 (36.2%) compared to 34 (72.3%) with FR 200 ml/s (p=0.001). In the 48 patients with LVEF 50%, amongst SV, FR and LVEF changes, only the latter was an independent predictor of death (HR=0.92, 95% CI=0.87–0.98, p=0.02) (Table 2). LVEF change of <5% was the best cut-off for the prediction of death (log rank p=0.004) and therefore for determination of

Abstract 124 Table 1 Patient echocardiographic characteristics

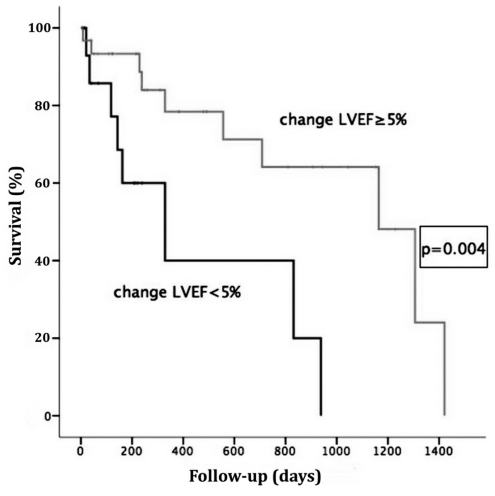
	Rest	Stress	p
HR (bpm)	74.9±14.5	97.5±18.7	<0.001
LVEF (%)	43±15.7	53.5±18.5	<0.001
AVA (cm^2)	0.77±0.13	0.92±0.2	<0.001
AVMG (mmHg)	25.7±6.7	35.3±10.9	<0.001
AVVmax (cm/sec)	328.1±43.3	384.8±52.5	<0.001
SV (ml)	56.6±14.1	64.4±16	<0.001
SVi (ml/m^2)	32.3±8.2	36.8±9.7	<0.001
Flow Rate (ml/sec)	179.8±34.6	240.1±55.4	<0.001

Abstract 124 Table 2 Univariable and multivariable analysis for prediction of all-cause mortality in patients with LVEF 50%

	Univariable			Multivariable		
	HR	95% CI	p value	HR	95% CI	p value
Age	1.002	0.9–1.1	0.96			
Hypertension	0.9	0.3–2.5	0.85			
Diabetes	0.9	0.3–2.6	**0.88**			
Presence of ischaemia on stress echo	2.68	0.59–12.14	0.20			
Change SV (ml)	0.99	0.9–1.04	0.67			
Change FR (ml/sec)	0.99	0.98–1.004	0.19			
Change LVEF (%)	0.92	0.87–0.98	0.02	0.92	0.87–0.98	0.02



Abstract 124 Figure 1



Abstract 124 Figure 2

CR (Figures 1-2). Increase in LVEF 5% had a significant impact on survival both on patients that underwent aortic valve intervention (log rank p=0.03) and those who underwent medical management (log rank p=0.01), as opposed to presence of SVFR (log rank p=0.234 and p=0.708 respectively).

Conclusions During LDDSE in LFLGAS normalised FR, not SVFR, is a better determinant of TSAS, whereas assessment of LVEF change instead of SVFR determines CR.

Valve Disease/Pericardial Disease/ Cardiomyopathy

125 EVALUATION OF TITIN CARDIOMYOPATHY IN PATIENTS WITH DILATED CARDIOMYOPATHY REVEALS A BLUNTED HYPERTROPHIC RESPONSE, AN EARLY ARRHYTHMIC RISK AND A SIGNIFICANT INTERACTION WITH ALCOHOL

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Background Titin truncating variants (TTNtv), found in~10%–20% of dilated cardiomyopathy (DCM), are notable for variable penetrance and expressivity. We evaluated whether TTNtv DCM patients had distinct phenotypic features, which may influence disease outcomes.

Methods Prospectively recruited DCM patients underwent comprehensive clinical evaluation, cardiac MRI and TTN sequencing.

Results Overall, 572 subjects, 388 men (67.8%), mean age 53.5±14.4 years, were recruited. TTNtv were found in 56 patients (9.8%) and were associated with lower indexed LV mass (LVMi) and thinner LV walls, in the absence of differences in LV volumes after adjusting for clinical covariates (LVMi 83.1 vs. 94.0g/m^2, p=0.008; max. LV wall thickness 9.1 vs. 10.1 mm, p=0.003; indexed LV end diastolic volume 122.7 vs. 131.3mls/m^2, p=0.07).

196/572 patients (34%) had atrial fibrillation or ventricular arrhythmia at recruitment. Adjusting for age, gender, baseline ventricular function, and left atrial volume, TTNtv independently associated with early arrhythmic burden (adjusted OR 2.90, CI 1.48 to 5.77, p=0.002). On sensitivity analysis, this association remained significant after exclusion of 12 patients with rare LMNA variants (adjusted OR 2.88, CI 1.45 to 5.81, p=0.003).

TTNtv alone did not predict LVEF but in the presence of a history of alcohol excess, LVEF was reduced by 17.5% (p<0.0001), independently of other predictors of LVEF (age, gender, NYHA class, mid-wall fibrosis, and a family history of NIDCM).

Conclusions These data demonstrate that DCM due to TTNtv is associated with a blunted hypertrophic response, highlighting possible disease mechanisms. We also demonstrate that TTNtv are independently predictive of early arrhythmia and show a significant gene-environmental interaction between TTNtv and alcohol, which may inform risk stratification.

126 DOES HYPERTROPHIC CARDIOMYOPATHY GENOTYPE AFFECT TISSUE DOPPLER IMAGING PARAMETERS OVER 3-YEAR FOLLOW-UP PERIOD?

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Introduction Recent studies suggest that tissue Doppler imaging (TDI) have prognostic value in hypertrophic cardiomyopathy (HCM). We aimed to identify if there was a difference in rate of deterioration of TDI values of diastolic function and longitudinal systolic function according to genotype status.

Methods Aprospective, single-centre observational study over 33 months was undertaken. Twenty-six HCM patients were assigned a group according to genotype status (G+ve (n=14) vs G-ve (n=12)). Differences in baseline and follow-up TDI

Abstract 126 Table 1 Changes in TDI parameters according to HCM genotype

Value		Baseline, mean (SD)	Follow up, mean (SD)	Change	p value for Change (G+ve vs G-ve)
Medial E	G+ve	6.33 (0.50)	7.85 (0.75)	+1.52	**0.002**
	G-ve	6.57 (0.48)	5.06 (0.46)	−1.51	
Lateral E	G+ve	9.68 (0.84)	9.86 (0.84)	+0.18	**0.034**
	G-ve	9.13 (0.73)	6.94 (0.73)	−2.19	
Average E	G+ve	8.00 (0.61)	8.86 (0.75)	+0.86	**0.003**
	G-ve	7.85 (0.53)	6.00 (0.50)	−1.85	
Medial S	G+ve	7.57 (0.48)	8.11 (0.69)	+0.54	0.200
	G-ve	7.19 (0.50)	6.68 (0.34)	−0.51	
Lateral S	G+ve	9.89 (1.01)	8.65 (0.61)	−1.24	0.387
	G-ve	9.15 (0.62)	7.76 (0.43)	−1.39	
Medial E/E	G+ve	12.59 (1.66)	9.73 (1.07)	−2.86	**0.004**
	G-ve	14.98 (2.09)	18.00 (2.58)	+3.02	
Lateral E/E	G+ve	8.33 (1.04)	7.23 (0.79)	−1.10	0.0003
	G-ve	9.98 (1.31)	14.05 (2.25)	+4.07	
Average E/E	G+ve	10.46 (1.31)	8.48 (0.87)	−1.98	**0.0002**
	G-ve	12.48 (1.66)	16.02 (2.30)	+3.54	