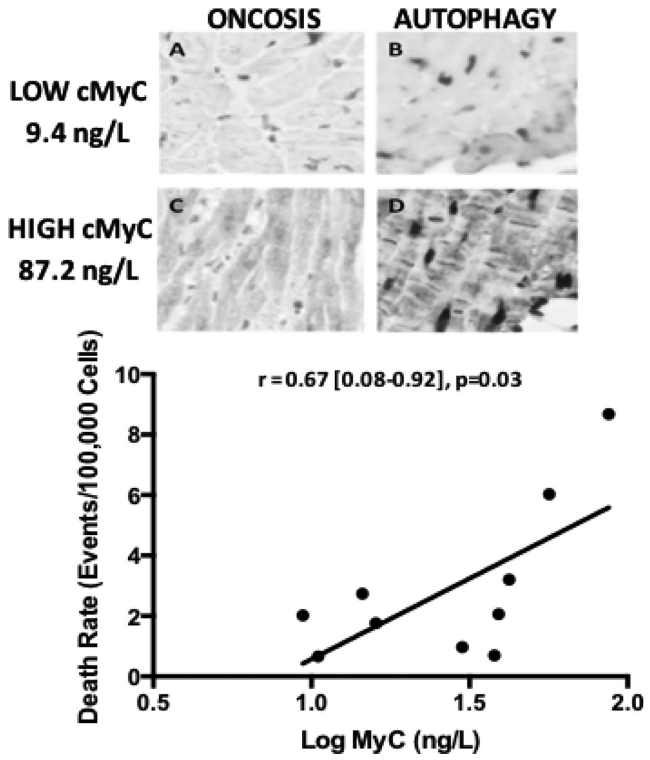

Abstract 151 Figure 2 *Oncosis* and *autophagy* in biopsy samples from patients with low and high cMyC concentrations, visualised using a 3,3′-diaminobenzidine staining. Plot shows correlation between cMyC and total myocyte death rate acros 10 patients tested

cohort who underwent aortic valve replacement. These samples underwent staining and analysis for myocyte death.

Results In the mechanism cohort, cMyC concentration correlated with left ventricular mass (r = 0.59, 95% CI 0.47–0.68, p < 0.0001), fibrosis volume (r = 0.57, 95% CI 0.45–0.67, p < 0.0001) and extracellular volume (r = 0.28, 95% CI 0.12–0.42, p = 0.0004) in patients with aortic stenosis but not in controls (Figure 1). In those with late gadolinium enhancement (LGE), cMyC was higher (32 [21–56] ng/L vs 17 [12–24] ng/L without LGE, p < 0.001). cMyC was unrelated to aortic transvalvular gradient or objective assessment of coronary disease. Unadjusted Cox proportional hazards analysis in the outcomes cohort showed greater all-cause mortality (HR 1.53 per doubling of cMyC, 95% CI 1.13–2.06, p = 0.006). In exploratory analyses, a relationship was observed between cMyC and the rate of myocyte death (expressed as the sum of apoptosis, oncosis and autophagy counts) in biopsy samples (r = 0.67, 95% CI 0.08–0.92, p = 0.03). Clear differences were observed in staining patterns for oncosis and autophagy between subjects with low and high cMyC concentrations (Figure 2).

Conclusion Serum cMyC concentration is associated with myocardial hypertrophy, fibrosis and an increased risk of mortality in aortic stenosis. The quantification of serum sarcomeric protein concentrations have major potential to provide objective measures of disease progression and to guide clinical decisions in patients with aortic stenosis.

152 EXPLORATORY ASSESSEMENT OF SIMULTANEOUS LEFT VENTRICULAR STRAIN AND VOLUME IN CHRONIC SEVERE AORTIC VALVE DISEASE

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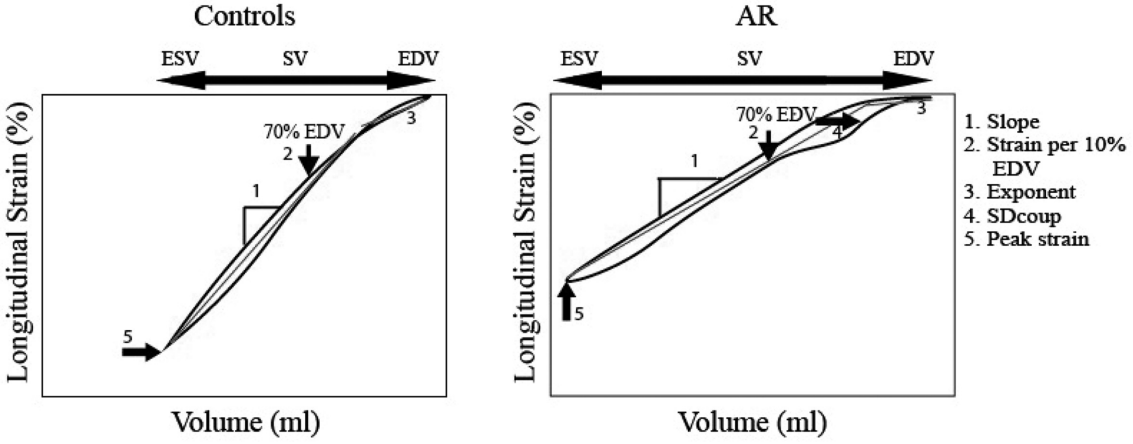
Introduction Aortic valve regurgitation (AR) and stenosis (AS) increase the workload of the left ventricle (LV), resulting in remodeling of chamber volume and walls that normalise wall stress. Whether functional characteristics of the heart, such as strain (ϵ), adapt as the heart remodels is unclear. Likewise, we know little about the relationship between changes in volume and strain across the cardiac cycle. This exploratory study utilised a novel technique (ϵ-volume loops) to establish the contribution of longitudinal (Lϵ) and transverse strain (Tϵ) to volume change throughout the cardiac cycle in patients with severe aortic valve disease.

Methods 27 participants with preserved ejection fraction (EF) were included consisting of AR (n = 7), AS (n = 10) and healthy controls (n = 10). Standard transthoracic echocardiography was used to obtain an apical 4 chamber image of the LV. Temporal Lϵ and Tϵ values were exported and divided into 5%-time increments. LV volumes were traced at each 5%-time increment. The ϵ-volume relationship was assessed by 1) the slope of the linear regression line in systole (Sslope) and diastole (Dslope), 2) polynomial equations to derive



Abstract 152 Figure 1 A schematic view of the methods used to asses the ϵ-volume loops. Left: a curve of a typical control ϵ-volume loop. Right: a curve of the ϵ-volume loop of a patient with AR

Abstract 152 Table 1 Characteristics Lϵ

Longitudinal Strain	Controls	AS	AR	P-value
Sslope (%/ml)	0.35 ± 0.06	0.26 ± 0.07*,†	0.12 ± 0.02*,†	<0.01
Dslope (%/ml)	0.33 ± 0.08	0.23 ± 0.08*,†	0.12 ± 0.02*,†	<0.01
Lϵ (%)	-19.5 ± 3.8	-16.7 ± 1.3*	-15.0 ± 1.9*	<0.01
SExp	0.95 ± 2.12	2.29 ± 2.25	3.34 ± 1.72	0.79
DExp	2.53 ± 2.56	0.69 ± 2.33	1.04 ± 2.18	0.22
SDcoup 90%	0.54 ± 1.58	1.61 ± 1.90	2.01 ± 2.24	0.26
SDcoup 80%	-0.28 ± 2.20	1.75 ± 2.34	2.34 ± 2.14	0.05
SDcoup 70%	-0.75 ± 2.32	1.60 ± 2.45*	2.28 ± 1.94*	0.02
SDcoup 60%	-0.88 ± 1.95	1.15 ± 2.14*	1.82 ± 1.55*	0.02
SDcoup 50%	-0.68 ± 1.46	0.40 ± 1.48	0.97 ± 1.06	0.06
SDcoup 40%	-0.14 ± 2.20	-0.64 ± 1.12	-0.28 ± 1.11	0.77

* = Significantly different from controls. † = Significantly different between AS and AR.

Abstract 152 Table 2 Characteristics Tϵ

Transverse Strain	Controls	AS	AR	P-value
Sslope (%/ml)	-0.19 ± 0.06	-0.26 ± 0.10†	-0.14 ± 0.04†	0.01
Dslope (%/ml)	-0.16 ± 0.06	-0.26 ± 0.11*,†	-0.13 ± 0.03†	<0.01
Tϵ (%)	9.9 ± 3.0	17.2 ± 5.6*	17.5 ± 6.5*	<0.01
SExp	1.98 ± 3.34	3.73 ± 5.00	4.10 ± 5.67	0.67
DExp	0.04 ± 7.57	3.34 ± 4.27	3.29 ± 3.67	0.69
SDcoup 90%	-1.25 ± 2.86	-0.25 ± 2.57	-0.40 ± 2.33	0.73
SDcoup 80%	-1.21 ± 3.40	0.06 ± 3.41	-0.52 ± 2.92	0.76
SDcoup 70%	-0.96 ± 3.64	0.27 ± 3.63	-0.62 ± 3.19	0.65
SDcoup 60%	-0.49 ± 3.54	0.37 ± 3.20	-0.69 ± 2.67	0.30
SDcoup 50%	0.20 ± 3.24	0.35 ± 2.24	-0.74 ± 1.39	0.59
SDcoup 40%	1.10 ± 3.19	0.23 ± 1.80	-0.76 ± 1.64	0.36

* = Significantly different from controls. † = Significantly different between AS and AR.

absolute ϵ values for% of end diastolic volumes (EDV) in the working physiological range of 10 to 60% EF, 3) a systolic and diastolic exponent (SExp and DExp) as determined by the difference between ϵ at 10% increments relative to ϵ at 60% EF and 4) systolic-diastolic coupling (SDcoup) as the difference between systolic and diastolic ϵ at each 10% increment of EDV (see Figure 1).

Results Patients with AS or AR had significantly lower peak Lϵ, higher peak Tϵ and a "flatter" longitudinal Sslope and Dslope compared to controls (see Tables 1 and 2). Following polynomial calculation to specific EDV these differences were partially normalised, suggesting the rate of change is, in part, related to chamber size. Lϵ but not Tϵ systolic-diastolic coupling was reduced in AS and AR compared to controls. There was a trend highlighting a higher SExp and DExp (indicating a reduction of ϵ contribution to volume change in early systole/late diastole) in both patient groups compared to controls for Lϵ and Tϵ. There was no significant difference in peak Lϵ and Tϵ between AS and AR patients but steeper slope values were observed in AS patients.

Conclusion This exploratory study shows the traditional shift in LV volumes between controls, AS and AR, but also indicates lower peak Lϵ and higher peak Tϵ values in AS and AR compared to controls. Although peak Lϵ or Tϵ were not different between AS and AR, we reported a distinct relationship between ϵ and volume between pathologies. Therefore, this novel technique may help to distinguish functional consequences of AS and AR and may have clinical relevance.

153 PREVALENCE OF LOW FLOW AND OF ABNORMAL LV LOADING METRICS AFTER AORTIC VALVE REPLACEMENT – A PRELIMINARY ECHOCARDIOGRAPHIC STUDY

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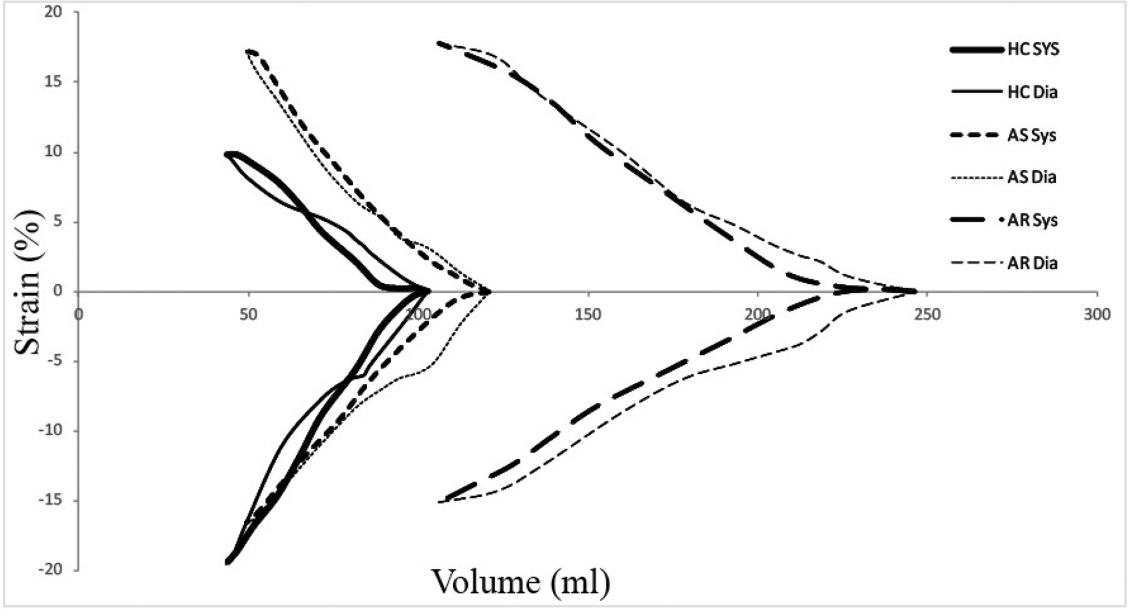
Background Since the report in 2007 of paradoxical low-flow aortic stenosis (AS) as a distinct entity there has been a lot of interest in low-flow states in patients with AS and preserved LV ejection fraction, their relation with indices of vascular function and in their effect on the conventional metrics used for characterising (AS). Little is known though on the



Abstract 152 Figure 2 Mean longitudinal and transverse ϵ-volume loops in controls, AS and AR patients respectively