Is there a relationship between LAVi and routine ECG parameters in hypertrophic cardiomyopathy?

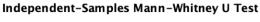
Background Pilot project informing statistical power, assessing the relationship between left atrial volume and ECG abnormalities in hypertrophic cardiomyopathy

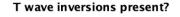
Method

- 90 consecutive adult patients derived from Freeman Hospital echo database between 8/18 to 04/23
- Baseline characteristics in figure 1
- ECGs assessed manually by one investigator (QRSd to nearest 5ms)
- Atrial dysrrhythmia defined as AF, AFL, and atrial pacing
- Sinus rhythm includes atrial sensing
- TWI not considered if in III, aVR and V1

Age median [IQR]	64 [50.1-75]
Sex male% (n)	61.1 (55)
BMI median [IQR]	28.4 [25.3-32.4]
LAVi available yes% (n)	80 (72)
LAVi median [IQR] ml ³ m ⁻²	43 [27.8-63]
ECG available yes% (n)	63.3 (57)
QRS duration median [IQR] ms	85 [80-120]
Atrial rhythm % (n)	SR, 77.2 (44);
	Atrial dysrrhythmia, 22.8 (13)
Ventricular conduction % (n)	Normal, 70.2 (40); NSIVCD, 5.3 (3);
	RBBB, 14 (8); LBBB, 1.8 (1); VP, 1.8 (1)
T wave inversion % (n)	45.3 (26)
Pathologic Q waves % (n)	12.3 (7)

Figure 1: Baseline population characteristics





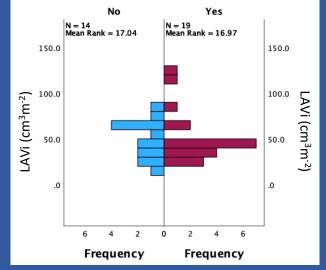


Figure 2: No association between LAVi and TWI



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Results

- LAVi not normally distributed
- Correlation between QRSd and LAVi found NS [R=-0.099, P=0.266]
- Association between presence of TWI and LAVi by Mann-Whitney U test (figure 2) found NS [p=0.986]
- Logistic regression all NS: TWI alone; TWI + QRSd; TWI + QRSd + age + BMI

Conclusions

- No statistically significant associations found between LAVi, QRSd and TWI Inadequate statistical power for subgroup analysis (e.g. localising TWI)
- We're planning a larger study with subgroup analysis, using E/A and strain (more specific for diastolic dysfunction)
- Limitations are small sample size, no formal sample method and no subgroup analysis