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Changes in Ventriculo-arterial coupling and Valvulo-arterial impedance in patients with Transcatheter Aortic Valve Implantation for Severe Aortic Stenosis

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BACKGROUND

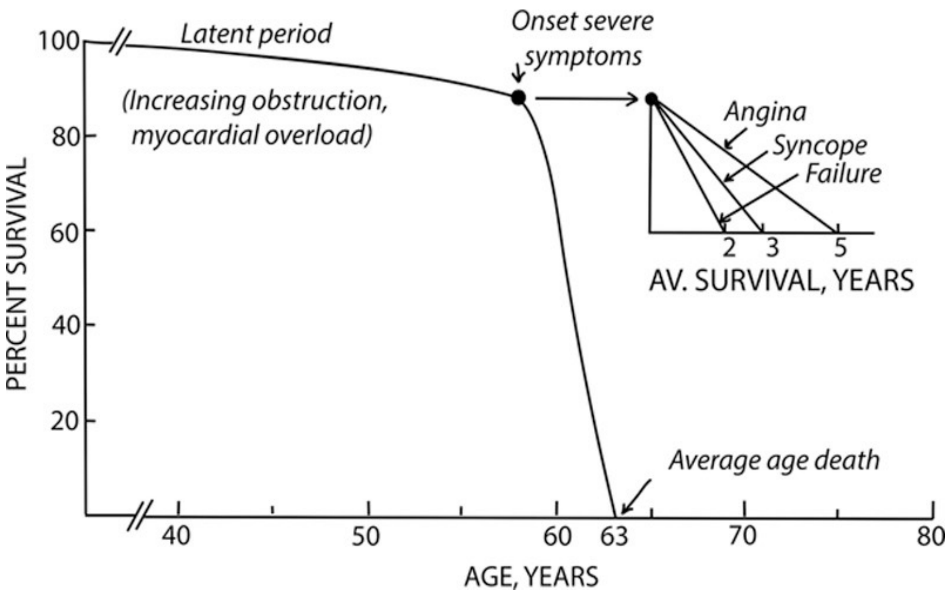
Left Ventricle in Severe Aortic Stenosis

Normal LV → Compensatory concentric hypertrophy → Fibrosis → Heart failure symptoms

Severe AS

Fibrosis:
Subendocardial ischemia
Mechanical Stress
Myofibroblast infiltration
Extracellular matrix expansion
Myocyte cell death

Heart failure symptoms:
Systolic dysfunction
Diastolic dysfunction



Ross J, Braunwald E. Aortic stenosis. **Circulation**. 1968; *37/38*(suppl V): V-61–V-

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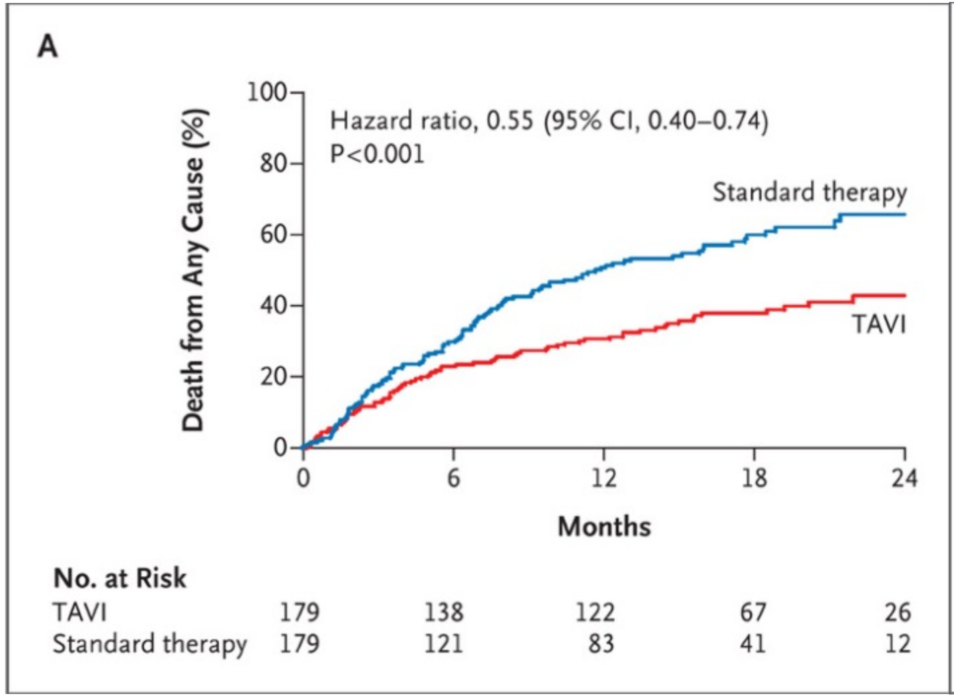
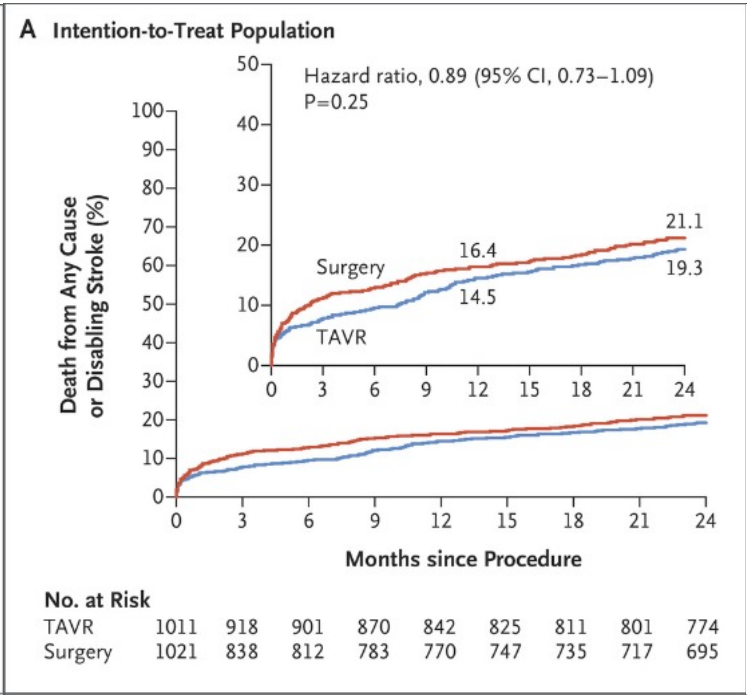
TAVI for AS

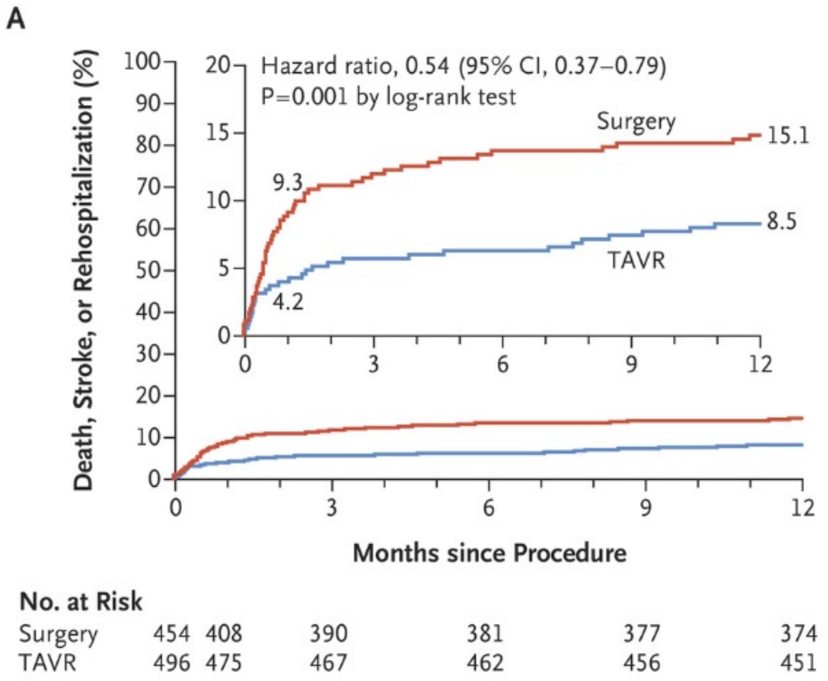
- Aortic valve replacement has tempered this outcome
- Transcatheter aortic valve replacement (TAVI) has emerged over the past 10-15 years as a good option for many patients



(Smith et al, 2011)



(Leon et al, 2016)



(Mack et al, 2016)

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AVR and LV function in Severe AS

1	A	1. In adults with severe high-gradient AS (Stage D1) and symptoms of exertional dyspnea, HF, angina, syncope, or presyncope by history or on exercise testing, AVR is indicated.[1–7]
1	B-NR	2. In asymptomatic patients with severe AS and an LVEF <50% (Stage C2), AVR is indicated.[8–11]

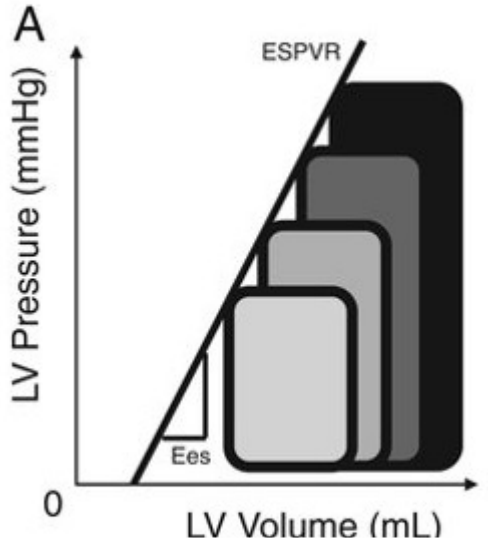
Otto et al., 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease. Circulation. 2021;143:e72–e227.

Ventriculo-Arterial Coupling (VAC) and Valvulo-Arterial Impedance (Zva)

VAC $\approx \frac{Ea}{Ees}$

(Normal 0.5 - 1.0)

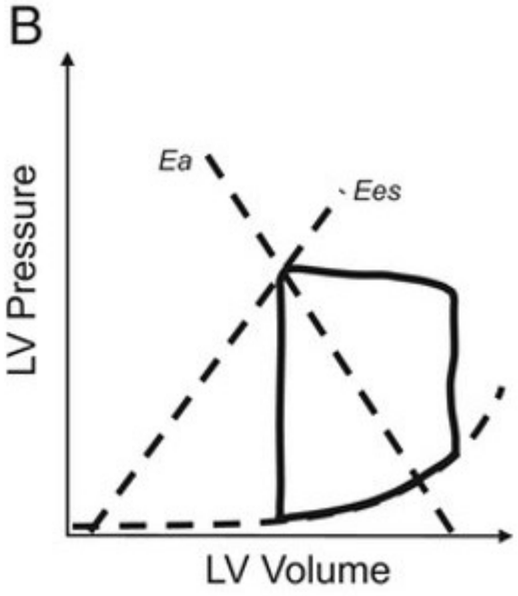
End-Systolic Elastance (Ees)
Load independent measure of contractility



$$E_{es(sb)} = [P_d - (E_{Nd(est)} \times P_s \times 0.9)]/[SV \times E_{Nd(est)}]$$

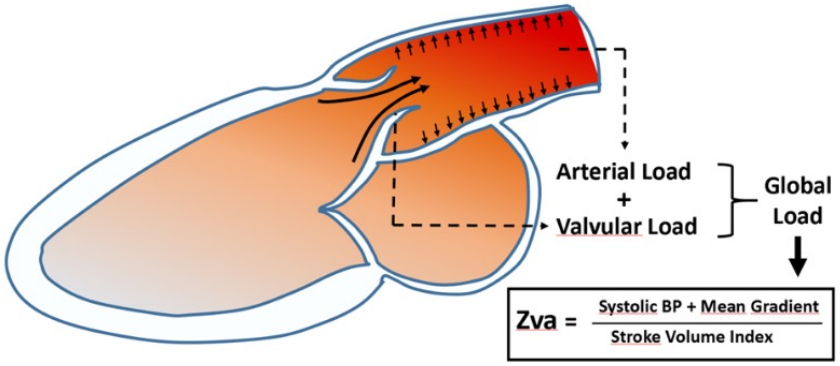
(Chen et al, 2001)

Effective Arterial Elastance (Ea)
Index of Afterload



Ea = End-systolic pressure/ Stroke volume
= (SBP x 0.9) / Stroke Volume

Zva



Estimate of global LV hemodynamic load, made up of

- Stenosis severity
- Volume flow rate
- Body Size
- Systemic Vascular resistance

Low: Zva ≤3.5
Moderate: 3.5 < Zva <4.5
High: Zva ≥ 4.5

(Hachicha et al, 2009)

Rationale and Objectives

Rationale: Describing how the LV and systemic vasculature interact with eachother in severe AS may better identify patients how would benefit from TAVI

Objectives: Describe the change in VAC and Zva pre-and-post TAVI in patients with severe aortic stenosis

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DESIGN AND METHODS

Design and Methods

Design:

- Retrospective cohort study

Methods:

- 100 patients randomly chosen from pre-existing database of all TAVIs done at UK Healthcare between 2012 and 2021
- Transthoracic echocardiograms (pre-TAVI and 1-3 month post-TAVI follow up), and TAVI CTs reviewed
- Zva and VAC calculated using previously validated non-invasive techniques

Statistics:

- The mean values for each of these measurements were compared using a paired T-test
- A p-value of ≤ 0.05 was chosen to characterize a significant difference in mean values for each variable measured.

Systolic/Diastolic Blood Pressure – At time of echocardiogram

Mean Gradient – across aortic valve

- If Atrial fibrillation, average of 3 measurements taken

Stroke Volume – calculated using the LVOT-VTI method

- LVOT diameter measured on pre-TAVR CT scan
- VTI of flow calculated through LVOT on TTE
- If atrial fibrillation, average of at least 3 measurements

Cardiac Output = $SV_{(\text{LVOT VTI Method})} \times HR_{(\text{at time of LVOT-VTI measurement})}$

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RESULTS

RESULTS

	Pre (N=100)	Post (N=100)	P-value
LVEF			
Mean (SD)	0.581 (0.138)	0.564 (0.145)	0.196
Median [Q1, Q3]	0.580 [0.501, 0.659]	0.576 [0.451, 0.676]	
Ees			
Mean (SD)	1.76 (0.674)	1.49 (0.688)	<0.001
Median [Q1, Q3]	1.66 [1.27, 2.18]	1.38 [0.933, 1.92]	
Ea			
Mean (SD)	1.65 (0.519)	1.80 (0.792)	0.0229
Median [Q1, Q3]	1.58 [1.28, 1.99]	1.70 [1.40, 1.98]	
Ea/Ees			
Mean (SD)	1.02 (0.387)	1.42 (0.810)	<0.001
Median [Q1, Q3]	0.927 [0.764, 1.24]	1.16 [0.955, 1.65]	
Cardiac Output			
Mean (SD)	5.31 (1.60)	5.47 (1.91)	0.358
Median [Q1, Q3]	4.98 [4.30, 6.31]	5.06 [4.31, 6.48]	
MAP			
Mean (SD)	91.1 (12.5)	94.8 (11.8)	0.0158
Median [Q1, Q3]	89.8 [83.0, 98.8]	95.1 [86.6, 102]	
SV			
Mean (SD)	52.6 (21.3)	51.4 (20.2)	0.589
Median [Q1, Q3]	46.9 [38.5, 64.1]	49.4 [35.2, 65.3]	

	Pre (N=100)	Post (N=100)	P-value
SVR			
Mean (SD)	1410 (456)	1490 (619)	0.131
Median [Q1, Q3]	1340 [1130, 1640]	1400 [1100, 1680]	
SBP			
Mean (SD)	133 (19.4)	144 (23.5)	<0.001
Median [Q1, Q3]	134 [117, 146]	141 [129, 160]	
MG			
Mean (SD)	47.7 (13.5)	10.7 (4.59)	<0.001
Median [Q1, Q3]	45.0 [40.9, 51.3]	10.0 [8.00, 13.3]	
ZVA			
Mean (SD)	4.89 (1.48)	4.19 (1.81)	<0.001
Median [Q1, Q3]	4.70 [3.80, 5.71]	3.77 [3.11, 4.84]	

LVEF: Left ventricular ejection fraction. **Ees:** End-systolic elastance. **Ea:** Arterial elastance. **MAP:** Mean arterial pressure. **SV:** Stroke Volume. **SVR:** Systemic vascular resistance. **SBP:** sytolic blood pressure. **MG:** mean aortic valve gradient. **ZVa:** Valvulo-arterial impedance

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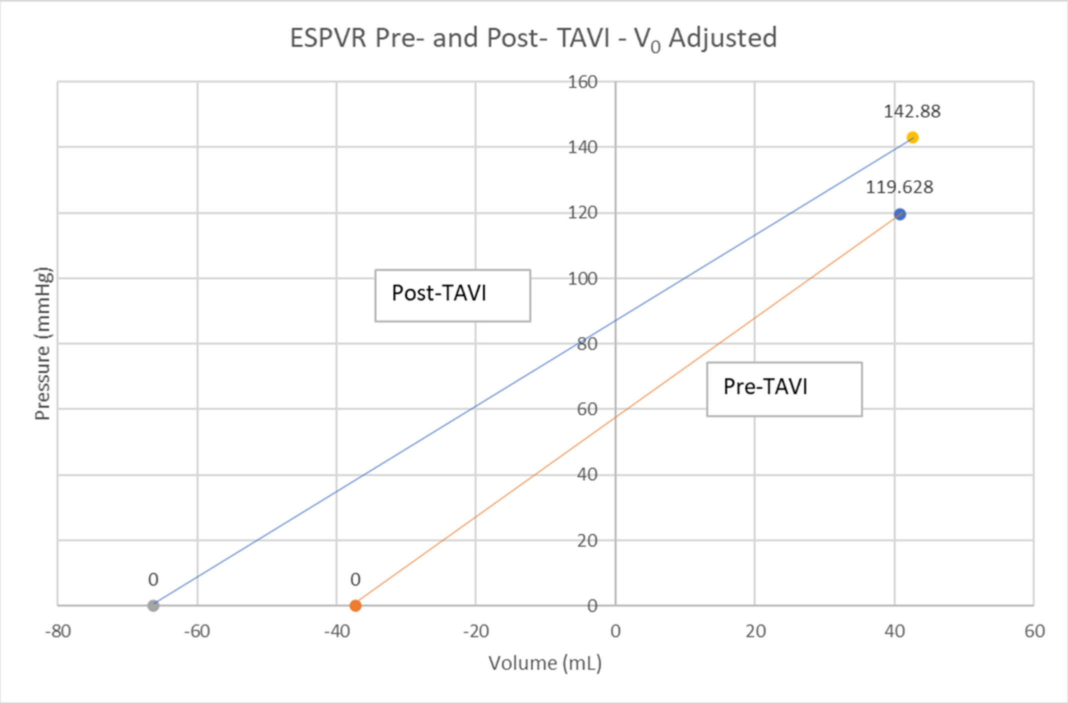
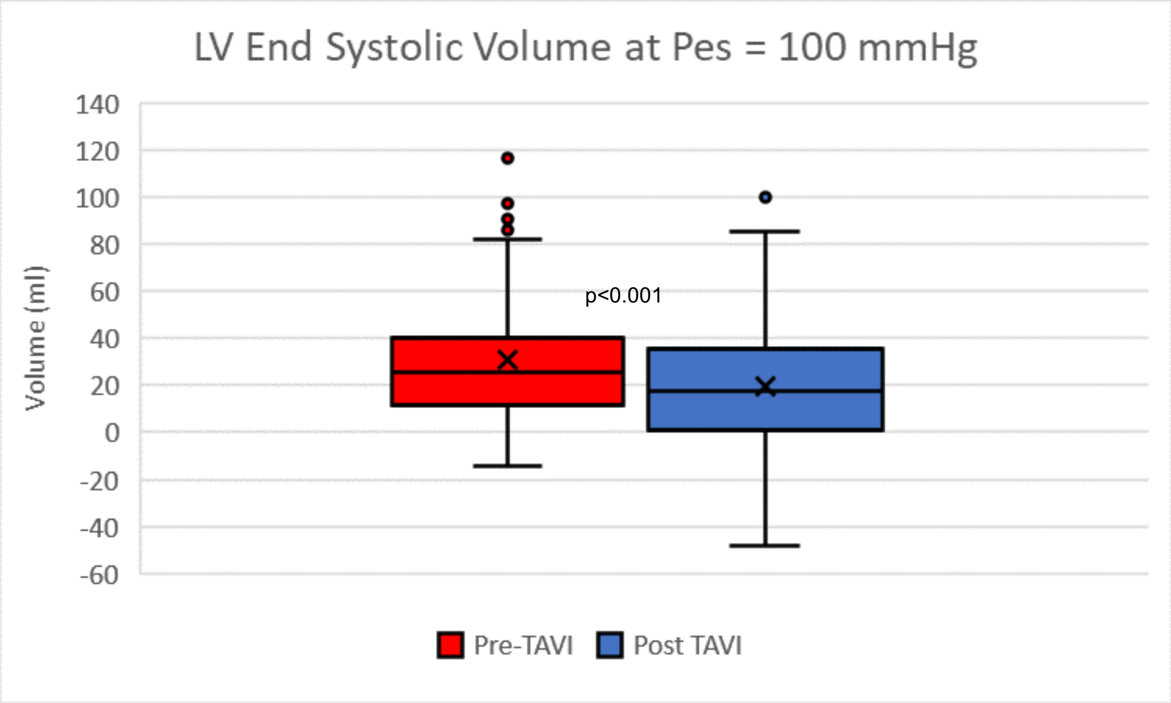
Post-TAVI Improvement in LV Contractile Function





ESPVR: End-systolic pressure-volume relationship
V0: volume-axis intercept of ESPVR (at P = 0 mmHg)

Pes: LV End-Systolic pressure

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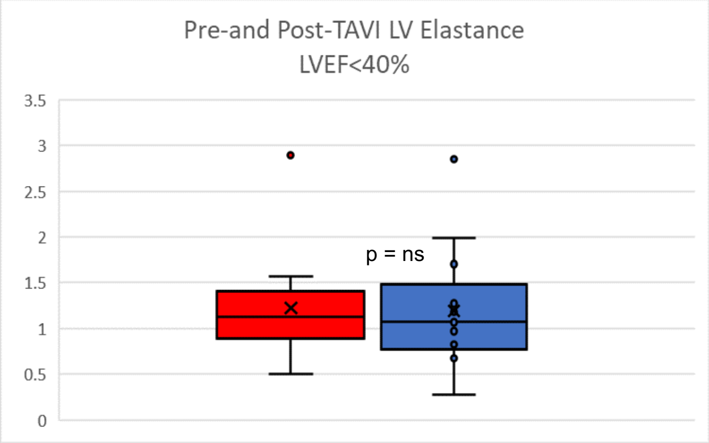
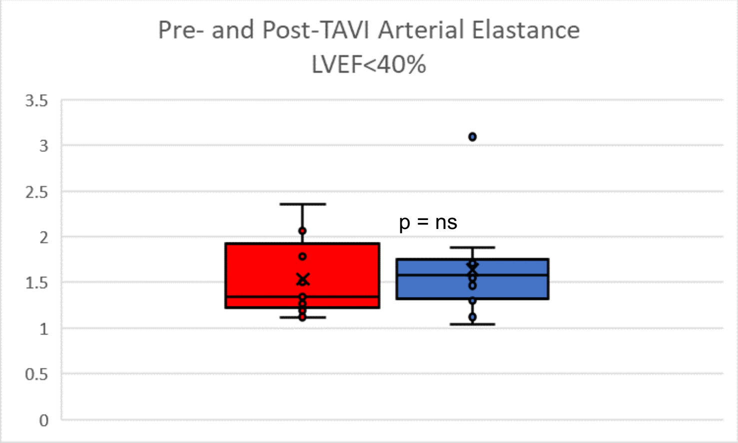
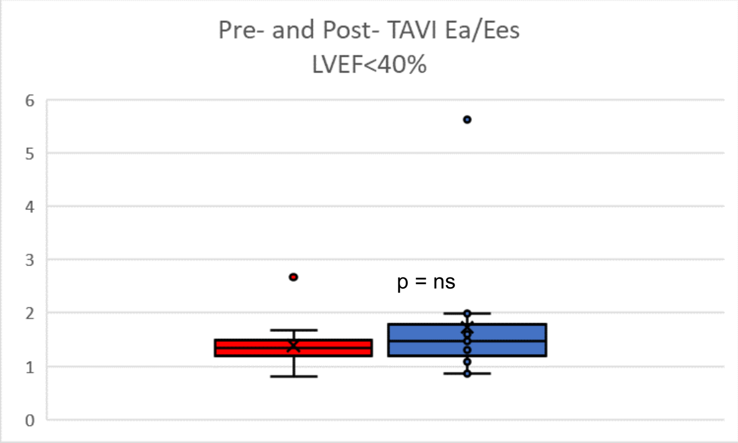
Relation of LV Ventricular Arterial Coupling to Pre-TAVI LVEF and Arterial Elastance







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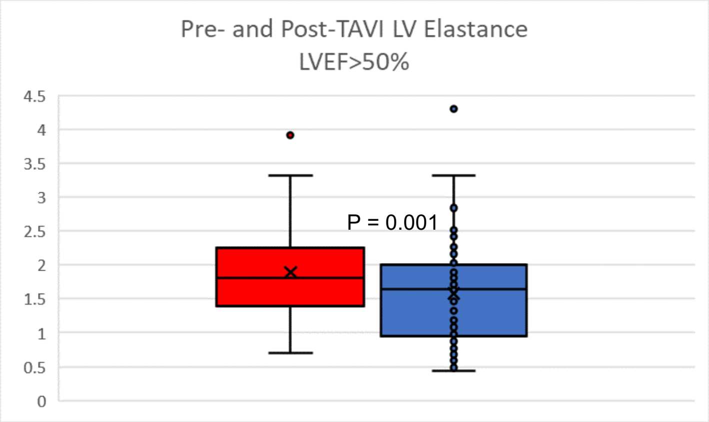

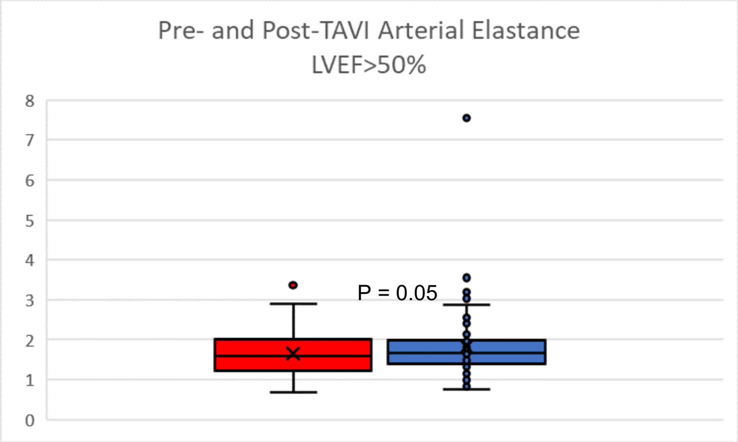

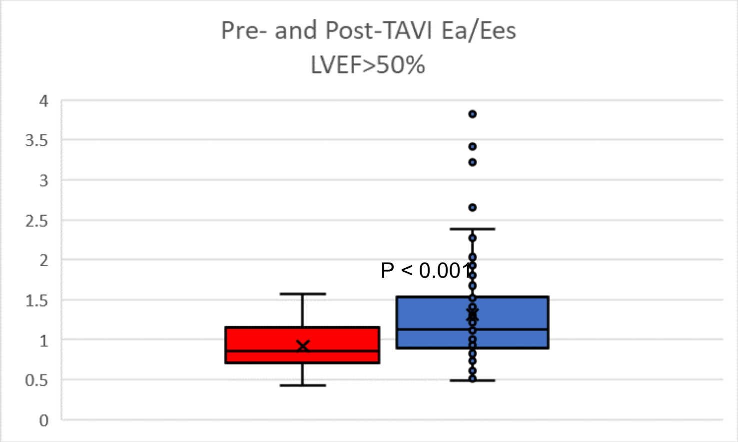

N = 76

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CONCLUSIONS

1) Non-invasive echocardiography was useful in demonstrating an improvement in LV contractile function following TAVI, using adjusted ESPVR and LVESV at Pes of 100 mmHg

2) However, LV elastance behaved differently, and decreased with an associated increase in arterial elastance leading to worsening LV-aorta coupling

- This suggests that higher arterial elastance adversely impacts LV elastance, highlighting the importance of optimizing BP control post TAVI
- Elevated systemic arterial stiffness from persistent hypertension likely tempers some of the benefit of TAVI

LIMITATIONS

- Small sample size
- Single center study
- Retrospective study

FUTURE INVESTIGATIONS

- Effect of comorbid conditions on changes in VAC and ZVa:
  - HTN
  - CAD
  - DM
  - Bundle branch block
  - Arrhythmias
  - Other valve disease

- Change in diastolic function following TAVI and its effect on VAC and Zva

- Effect of changes in Ea/Ees and Zva in clinical outcomes (rehospitalizations, mortality)

Questions?

Thanks to:

- Dr. Paul Anaya, MD, PhD – Principle Investigator (Gill Heart & Vascular Institute)
- Dr. Awa Drame – PGy-5, Cardiology (Gill Heart & Vascular Institute)

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