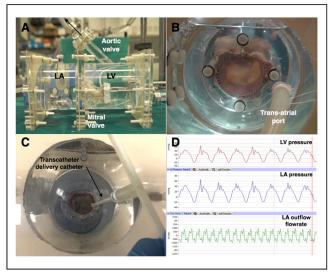
**BACKGROUND** Interest in transcatheter mitral valve repair/replacement (TMVR) for primary and secondary mitral regurgitation (pMR, sMR) is immense. Though several TMVR technologies are in development, many failed to achieve consistent reduction in MR and present with a risk of thrombosis from non-physiological hemodynamics. We report a novel bench model of pMR and sMR, to investigate the safety and efficacy of new TMVR devices.

**METHODS** An optically clear flow chamber with a left atrium (LA), left ventricle (LV), a bileaflet mechanical aortic valve, and a native pig mitral valve (Fig 1A, 1B). Physiological pressures were generated by a programmable pump connected to the LV, which determined mitral valve opening and closure (Fig 1D. pMR was induced by transecting one or more marginal chordae tendinae or moving the papillary muscle tips towards the annulus to induce bileaflet billowing. sMR was induced by displacing the papillary muscles away from the annulus and tethering the leaflets.

**RESULTS** Physiological transmitral pressure and flow enabled normal mitral valve closure and opening at baseline. Leaflet billowing or flail captured the human like pMR lesions with absolute control over the cusp involved and severity of billowing. Symmetric and asymmetric tethering could induce type I and type IIIb sMR lesions. A 24Fr MitraClip catheter was successfully inserted into the LA via the trans-atrial port, indicating use of this bench model for testing other transcatheter devices (Fig 1C).



**CONCLUSION** We report a robust bench top model to mimic human like mitral valve lesions to test the safety and efficacy of TMVR devices.

CATEGORIES STRUCTURAL: Valvular Disease: Mitral

## TCT-632

Predictive Value of Age-Adjusted Charlson Comorbidity Index for 1-Year, 3-Year and 5-Year Mortality in Patients Following Transcatheter Mitral Valve Repair



Mike Saji, <sup>1</sup> Marc Katz, <sup>2</sup> Gorav Ailawadi, <sup>3</sup> Dale Fowler, <sup>4</sup> Damien LaPar, <sup>5</sup> Leora Yarboro, <sup>6</sup> Ravi Ghanta, <sup>7</sup> John Kern, <sup>8</sup> John Dent, <sup>9</sup> Michael Ragosta, <sup>10</sup> Scott Lim<sup>11</sup>

<sup>1</sup>University of Virginia, Charlottesville, Virginia, United States; <sup>2</sup>Bon Secours Heart & Vascular Institute, Richmond, United States; <sup>3</sup>University of Virginia, Charlottesville, Virginia, United States; <sup>4</sup>Instituto de Cardiologia / Fundação Universitária de Cardiologia (IC/FUC); <sup>5</sup>Instituto de Cardiologia / Fundação Universitária de Cardiologia (IC/FUC); <sup>6</sup>University Hospital of Wales; <sup>7</sup>Cardiovascular Surgeon, Boston, Massachusetts, United States; <sup>8</sup>Instituto de Cardiologia / Fundação Universitária de Cardiologia (IC/FUC); <sup>9</sup>University of Virginia; <sup>10</sup>Unknown, Charlottesville, Virginia, United States; <sup>11</sup>UVA Medical Center, Charlottesville, Virginia, United States

**BACKGROUND** This study aimed to determine if age-adjusted Charlson comorbidity index could predict mortality in patients undergoing transcatheter mitral valve repair (TMVR), and to assess its discriminatory performance in long-term outcomes. Comorbidity increases markedly with aging, and they often negatively impact its prognosis. Although mortality with TMVR is significantly less than for open mitral valve surgery in this population, it remains a concern to identify which patients will benefit from this treatment. Some prognostic metrics have been reported to guide better patient selection, however,

universal risk stratification measures in this population, have not been established.

**METHODS** We retrospectively reviewed 222 patients undergoing TMVR. Cox proportional hazard models were applied to select the demographic characteristics that were associated with cumulative mortality. Receiver operating-characteristic analyses were performed for predicting all-cause mortality, and discriminatory performance was assessed.

**RESULTS** We found age-adjusted Charlson comorbidity index (hazard ratio 1.33, 95% confidence interval 1.16-1.51, p <0.001), New York Heart Association classification, atrial fibrillation were independently associated with mortality. The age-adjusted Charlson comorbidity index demonstrated excellent discriminative performance for predicting mortality at 3 and 5 years (area under the curve 0.81 and 0.83, respectively). They were greater than those of STS score and greatest in any other single parameters at 1, 3, and 5 years in ROC analysis. Kaplan-Meier curve demonstrated age-adjusted Charlson comorbidity index  $\geq$ 8 had poor prognosis following TMVR.

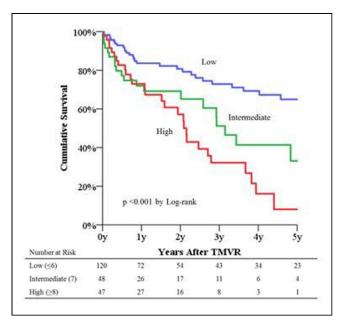


Table 3. Predictors of Mortality

	Univariate analysis		Multivariate analysis	
	HR	p Value	HR	p Value
Age-adjusted Charlson comorbidity index (per 1 point increase)	1.34	<0.001	1.33	<0.001
NYHA classification (per class increase)	1.54	0.007	1.41	0.04
Coronary artery disease	1.66	0.05		
Atrial fibrillation	1.93	0.008	2.05	0.004
Hemoglobin (per 1g/ dL decrease)	1.17	0.02		
LV systolic diameter (per 10 mm increase)	0.83	0.12		

HR = hazard ratio; CI = confidence interval; NYHA = New York Heart Association, LV = Left ventricle.

**CONCLUSION** The age-adjusted Charlson comorbidity index could predict mortality, and had an excellent discriminative performance for predicting longer-term outcomes in patients undergoing TMVR.

CATEGORIES STRUCTURAL: Valvular Disease: Mitral