allows the calculation of the effective AVA, but the accuracy of this measurement has been criticized and is not part of core guidelines. Multidetector computed tomography (MDCT) supports AS diagnosis using valvular calcium scoring and MDCT can delineate aortic cusps, but anatomical AVA measure remains untested.

**Purpose:** To compare the measurement of 4D-MDCT derived anatomical AVA, obtained with new, custom-made software, with effective AVA by transthoracic echocardiography (TTE) continuity equation.

**Methods:** Twenty patients with severe AS and clinically indicated 4D-MDCT of the aortic valve were included. AVA was obtained using continuity equation for Doppler-Echocardiography. Using 4D-MDCT with contrast after imaging registration, custom semi-automated software allowed aortic cusp delineation and anatomical AVA measurement. With this software, a systolic 3D model of the valve is obtained after cusps' profiling using 18 automatically generated long-axis planes (Figure, top panel). Then, orifice area (anatomical AVA) was automatically calculated using 3 different algorithms (Figure, bottom panel): by using smallest 2D-projection of aortic cusps profile (method A), by computing 2D area of cusps' free margin (blue), and by using any plane (yellow) with the smallest area between cusps (C). Results: In 18 out of 20 patients (80%) MDCT image quality allowed complete delineation of aortic cusps. AVA by Doppler-Echo was 82±15 mm². Anatomical AVA measured 80±16 mm² for method-A, 88±20 mm² for method-B, 93±21 mm² for method-C, and 87±19 mm² when averaging over the three methods. Absolute differences between Echo and 4D-MDCT measurements were 7.7±4.6 mm² (p for difference=0.3; r=0.85, p<0.001) for method-A; 9.2±6.8 mm² (p for difference=0.074; r=0.86, p<0.001) for methods-B; 11.9±9.1 mm² (p for difference=0.0002; r=0.90, p<0.0001) for method-C; and 7.7±5 mm² (p for difference=0.003; r=0.89, p<0.001) for the average. Analysis of regression slopes (>1 echo lower than MDCT) was observed for methods-B, -C, and -average (1.26, 1.13, and 1.11 respectively) but was 0.93 for method-A.

**Results:** 14 of the total 64 (22%) were excluded because of inadequate image quality. Analysis was performed on the remaining 50 patients (mean age 76.5±8.4 years, 58% male). 41 patients had severe AS with an AVA trad <1 cm². Mean LVO-3D resulted to be higher than mean LVO-3D trad (3.91±0.530 cm² vs. 2.98±0.689 cm², p<0.001). As expected, mean AVA3D was greater than AVA-trad (1.02±0.333 cm² vs. 0.78±0.260 cm², p<0.001). Both AVA-trad and AVA3D had a significant correlation with MPG (r=0.684 and r=0.739 respectively, p<0.001 for both). Among the 41 patients with severe AS by AVA-trad, according to AVA3D: 19/20 patients with NF-HG/LF-HG remained in the same category, 5/8 patients with LF-LG and 12/13 with NF-LG were reclassified as non severe AS.

**Conclusion:** 3D-TTE derived functional AVA allowed to reclassify AS severity in 81% (17/21) of patients with discrepant AVA-trad and MPG (LF-LG or NF-LG).

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**Left atrial dysfunction as a pathway to heart failure symptoms in patients with severe aortic stenosis and preserved left ventricular ejection fraction**

C.C. Beladan1, A. Calin1, A. Mateescu1, M. Rosca1, R. Enache1, S. Botetatsu1, N. Majina1, C. Calin1, C. Gheghina1, B.A. Popescu1,1 University of Medicine and Pharmacy “Carol Davila”, Eurocoelab, Bucharest, Romania; 2Institute of Emergency for Cardiovascular Diseases “Prof. Dr. C.C. Iliescu”, Bucharest, Romania.

**Background:** Although prognosis in asymptomatic patients (pts) with severe aortic stenosis (AS) is relatively benign, the risk increases abruptly with symptom occurrence. The relationship between left atrium (LA) dysfunction and heart failure (HF) symptoms has been demonstrated in several settings of left ventricular (LV) myocardial dysfunction such as HF with preserved LV ejection fraction (LVEF) or hypertrophic cardiomyopathy. However, data regarding the contribution of LA dysfunction to the patients’ symptoms in severe AS is scarce.

**Aim:** We aimed to evaluate the usefulness of LA function over other parameters related to the symptomatic status (eg BNP serum values, LV global longitudinal strain - GLS, LVEF), E/e ratio and LA size in stratifying the risk of pts with severe AS.

**Methods:** We prospectively enrolled 291 consecutive pts (66±11 yrs, 57% men) with severe AS (indexed aortic valve area, AVA <0.6 cm²/m²) and preserved LVEF (>50%), in sinus rhythm, with no more than mild aortic or mitral regurgitation. Pts were divided in two groups based on the presence of HF symptoms: symptomatic (238) or asymptomatic (53 pts). A negative exercise echocardiogram/ECG test was required to confirm the asymptomatic status. A comprehensive echocardiographic performance was evaluated using speckle tracking echocardiography.

**Results:** No significant differences were found between symptomatic and asymptomatic pts regarding age (66±10 vs 64±12 yrs), cardiovascular risk factors and comorbidities (ie smoking, hypertension, dyslipidemia, chronic kidney disease) (p>0.10 for all). Left ventricular EF and geometry - diameters, volumes, LV mass index (146±38 vs 146±38 g/m²), relative wall thickness and E/e’ average ratio (15±5 vs 13.5±4.8) were similar between groups (p>0.10 for all) despite a higher AAI in asymptotic pts (0.44 vs 0.39 cm²/m², p<0.001). Moreover, in symptomatic pts BNP values (in BNP: 5.4±1.0 vs 4.3±1.0, p<0.019), LAI (48±15 vs 42±15, p=0.033) and PAs (343 vs 30±8 mmHg, p=0.029) were significantly higher, whereas GLS (-14.6±3.6 vs -15.9±3.4%, p=0.026), peak LA longitudinal strain (17±7 vs 23±7%, p<0.001) and strain rate parameters (SSr, ESr, ASr, p<0.001) were significantly lower compared to asymptomatic pts. In the multivariable analysis, peak LA longitudinal strain was the only independent correlate of HF symptoms (p<0.011).

**Conclusions:** Peak LA longitudinal strain is an independent correlate of the presence of HF symptoms in patients with severe AS and preserved LVEF. A non-invasive echocardiographic evaluation of LA function would be useful to improve risk stratification in this clinical setting.