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# The Role of Index of Valvular-Arterial Impedance and Systemic Arterial Compliance after Aortic Valve Replacement

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Mechanical valvular obstruction and reduced arterial compliance combine to increase left ventricular afterload in patinets with aortic stenosis (AS).[1] As a result of the recognition that valvular and arterial abnormalities both play important roles in determining the overall impedance to left ventricular ejection in AS, it is now clear that standard methods of quantifying valvular stenosis, which focus entirely on the valve itself do not adequately characterize the severity, predict the onset, progression, and magnitude of symptoms, or identify the incidence of subsequent adverse event.[2-7]

The valvuloarterial impedance (Zva) provides an estimate of the global left ventricle (LV) hemodynamic load that results from the summation of the valvular and vascular loads, and the concept is very useful because it incorporates stenosis severity, volume flow rate, body size, and systemic vascular resistance. Moreover, Zva can easily be calculated using Doppler echocardiography from 3 simple measurements, that is, the systemic arterial compliance (SAC) in the LV outflow tract, the transvalvular mean gradient, and systolic arterial pressure, it is superior to the standard indexes of AS severity in predicting LV dysfunction. Zva is the best-suited and most relevant parameter to clinically quantify this "global or total" increase in LV hemodynamic load. There is few data regarding effects of surgical aortic valve replacement (AVR) on Zva and SAC.[8]

In patients with AS undergoing transcatheter aortic valve implantation (TAVI), acute declines in Zva were reported.[9] Reductions in Zva observed 1 month after TAVI also were shown to persist during a 2-year follow-up,[10] suggesting that early assessment of Zva may provide important intermediate-term prognostic information. SAC was unchanged concomitant with persistent hypertension and widened pulse pressure during 2-year follow-up after TAVI[10] because the chronic pathologic changes responsible for increased arterial stiffness with age are most likely irreversible despite treatment with antihypertensive and statin medications.

In this issue of the Journal of Cardiovascular Ultrasound, Jang et al.[11] tried to evaluate the relationship between Zva and the LV hypertrophy (LVH) regression after AVR and the physiologic role of Zva and SAC in severe AS. Authors reported Zva and SAC are major determinants of concentric remodeling in AS and LVH regression after AVR. Progressive decrease in SAC can partly explain incomplete LVH regression after AVR, which suggests that SAC could be a potential therapeutic target. Furthermore, these authors suggested that SAC could be used as a therapeutic target after AVR to obtain complete regression of LVH and yield better long-term outcomes.

In this study, the parameter of LVH was LV mass (LVM) index/LV end-diastolic volume (LVEDV) index. The measurement of LVM and LVEDV was based on the echocardiographic assessment. Currently, gold standard method of LVM and LVEDV is cardiac magnetic resonance imaging. Also, medial follow-up was quite short (2.4 years). Therefore, more data are clearly required in larger scaled population to determine the role of SAC as a therapeutic target after AVR. Comparison TAVI and AVR would be better understanding the pathophysiologic role of Zva and SAC.

In conclusion, although there were some limitation, the study by Jang et al.[11] demonstrates the relationship between

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Zva and the LVH regression after AVR and the physiologic role of Zva and SAC in severe AS.

## References


1. Briand M, Dumesnil JG, Kadem L, Tongue AG, Rieu R, Garcia D, Pibarot P. *Reduced systemic arterial compliance impacts significantly on left ventricular afterload and function in aortic stenosis: implications for diagnosis and treatment. J Am Coll Cardiol 2005;46:291-8.*
2. Hachicha Z, Dumesnil JG, Pibarot P. *Usefulness of the valvuloarterial impedance to predict adverse outcome in asymptomatic aortic stenosis. J Am Coll Cardiol 2009;54:1003-11.*
3. Lancellotti P, Donal E, Magne J, Moonen M, O'Connor K, Daubert JC, Pierard LA. *Risk stratification in asymptomatic moderate to severe aortic stenosis: the importance of the valvular, arterial and ventricular interplay. Heart 2010;96:1364-71.*
4. Maréchaux S, Carpentier E, Six-Carpentier M, Asseman P, LeJemtel TH, Jude B, Pibarot P, Ennezat PV. *Impact of valvuloarterial impedance on left ventricular longitudinal deformation in patients with aortic valve stenosis and preserved ejection fraction. Arch Cardiovasc Dis 2010; 103:227-35.*
5. Levy F, Luc Monin J, Rusinaru D, Petit-Eisenmann H, Lelguen C, Chauvel C, Adams C, Metz D, Leleu F, Gueret P, Tribouilloy C. *Valvuloarterial impedance does not improve risk stratification in low-ejection fraction, low-gradient aortic stenosis: results from a multicentre study. Eur J Echocardiogr 2011;12:358-63.*
6. Zito C, Salvia J, Cusmà-Piccione M, Antonini-Canterin F, Lentini S, Oreto G, Di Bella G, Montericcio V, Carerj S. *Prognostic significance of valvuloarterial impedance and left ventricular longitudinal function in asymptomatic severe aortic stenosis involving three-cuspid valves. Am J Cardiol 2011;108:1463-9.*
7. Lancellotti P, Magne J, Donal E, Davin L, O'Connor K, Rosca M, Szymanski C, Cosyns B, Piérard LA. *Clinical outcome in asymptomatic severe aortic stenosis: insights from the new proposed aortic stenosis grading classification. J Am Coll Cardiol 2012;59:235-43.*
8. Pagel PS, Schroeder AR, De Vry DJ, Hudetz JA. *Aortic valve replacement reduces valvuloarterial impedance but does not affect systemic arterial compliance in elderly men with degenerative calcific trileaflet aortic valve stenosis. J Cardiothorac Vasc Anesth 2014;28:1540-4.*
9. Giannini C, Petronio AS, De Carlo M, Guarracino F, Benedetti G, Delle Donne MG, Dini FL, Marzilli M, Di Bello V. *The incremental value of valvuloarterial impedance in evaluating the results of transcatheter aortic valve implantation in symptomatic aortic stenosis. J Am Soc Echocardiogr 2012;25:444-53.*
10. Katsanos S, Yiu KH, Clavel MA, Rodés-Cabau J, Leong D, van der Kley F, Ajmone Marsan N, Bax JJ, Pibarot P, Delgado V. *Impact of valvuloarterial impedance on 2-year outcome of patients undergoing transcatheter aortic valve implantation. J Am Soc Echocardiogr 2013;26:691-8.*
11. Jang JY, Seo JS, Sun BJ, Kim DH, Song JM, Kang DH, Song JK. *Impact of valvuloarterial impedance on concentric remodeling in aortic stenosis and its regression after valve replacement. J Cardiovasc Ultrasound 2016;24: 201-7.*