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DISCUSSION FORUM

Discussion forum: it is time to assess left ventricular segmental remodelling in aortic stenosis

Jan Stassen [1,2] and Jeroen J Bax[1,3]*

[1]Department of Cardiology, Leiden University Medical Center, Albinusdreef 2, 2300 RC, Leiden, The Netherlands; [2]Department of Cardiology, Jessa Hospital Hasselt, Stadsomvaart 11, 3500 Hasselt, Belgium; and [3]Department of Cardiology, Turku Heart Center, University of Turku and Turku University Hospital, Kiinamyllynkatu 4-8, FI-20520, Turku, Finland

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We thank Dr. Yalçin *et al.* for their interest in our study which evaluated the association between left ventricular (LV) hypertrophy and all-cause mortality in patients with moderate aortic stenosis (AS).[1] Left ventricular hypertrophy is an important predictor of cardiovascular morbidity and mortality in many cardiovascular diseases, including severe AS.[2] Recently, reduced survival has been reported in patients with moderate AS.[3] This observation implies that AS severity probably needs to be considered as a continuous variable, with each incremental increase in AS severity imposing an increased pressure load on the LV with consequent LV concentric remodelling. The results of our study indicate that LV concentric hypertrophy was frequently (36%) present in patients with moderate AS.[1] Because LV hypertrophy leads to LV myocardial fibrosis (which is strongly associated with mortality in patients with severe AS[4]) and occurs before a reduction in LV ejection fraction is observed, assessment of LV remodelling may have important prognostic implications in patients with moderate AS.

The concept that segmental remodelling could be an underlying mechanism in moderate AS and may have prognostic implications is of interest. Even though the mechanisms leading to basal hypertrophy in afterload-induced LV hypertrophy remain unclear, Laplace's law indicates that the larger the LV radius, the larger the wall tension that is required to withstand the LV pressure. Because the longitudinal fibres of the basal septum have the largest radius, they would experience the greatest component of wall stress. In addition, the incremental pressure load created by the right ventricular pressure exerts additional stress on the inter-ventricular septum, potentially explaining why the basal septum is more susceptible to develop LV hypertrophy in patients who already have an increased afterload due to moderate AS.[5] Other factors, such as delayed activation of the basal septum compared with other myocardial segments and a more developed sympathetic innervation at the basal septum, could also play a role. Despite these findings, strong outcome data regarding LV segmental remodelling in cardiovascular diseases are lacking. In addition, although segmental remodelling can differentiate between afterload-induced LV hypertrophy (i.e. ampulla-shaped morphology) and hypertrophic cardiomyopathy (i.e. catenoid morphology),[6] it does not discriminate between hypertrophy caused by increased *valvular* afterload versus increased *vascular* afterload. From a clinical point of view, discrimination between valvular-induced LV remodelling and vascular-induced LV remodelling may be even more important and could have important therapeutic implications in patients with moderate AS.

Selection of patients with moderate AS who perhaps may benefit from early aortic valve replacement remains challenging and assessment of LV remodelling is only one part of the evaluation. The recognition and treatment of associated comorbidities and optimization of LV heart failure should currently remain the first-line treatment. Although LV segmental remodelling is currently not routinely assessed in patients with AS, we agree with Dr. Yalçin *et al.* that this topic merits further investigation.

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* Corresponding author. Tel: +31 71 526 2020; Fax: +3171 526 6809, E-mail: j.j.bax@lumc.nl

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