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# Problem Is in the Subclinical Afterload Mismatch

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In the era of aging society, prevalence of degenerative aortic stenosis (AS) increases rapidly.[1] Systolic hypertension and widening of pulse pressure due to stiffened aorta are pathognomonic phenomenon in the elderly population.[2] Compared to previous decades, echocardiographic evaluation of aortic valve become more convenient, accordingly incidence of newly diagnosed AS has increased markedly even in asymptomatic individuals.[1] As degenerative AS is progressive disease in nature, monitoring by echocardiography provides crucial information in terms of decision making of surgical timing.

Important clinical issues were raised by Hachicha et al.[3] that increased afterload could make low-flow severe AS in spite of preserved left ventricular (LV) ejection fraction, which is mainly by lower stroke volume index due to small LV cavity accompanied by increased LV mass to volume ratio and lower midwall fractional shortening. Accompanied with these geometrical changes, higher blood pressure and lower aortic compliance could result in low-gradient AS. Finally these structural and functional changes induce low-flow and low-gradient AS even in preserved LV ejection fraction. Important clinical finding was that these patients were associated with poorer outcome.[3] Therefore, some concerns were raised about low-flow and low-gradient AS associated with high blood pressure and high global afterload. This is clinically important because currently trans-aortic valve pressure gradient and effective orifice area measured by continuous equation are choice of grading the AS severity. In addition, blood pressure changes time to time, especially in elderly individuals. Therefore if trans-aortic valve pressure gradient or effective orifice area changed significantly by blood pressure, it would be an important issue to be considered when monitoring the AS severity by echocardiography. To answer this question, Little et al.[4] conducted a novel study regarding "Impact of blood pressure on the Doppler echocardiographic assessment of AS" by using hand grip exercise and phenylephrine infusion. In that study, they concluded that acute blood pressure elevation due to increased systemic vascular resistance can affect the Doppler-echo evaluation of AS severity. However, the impact of blood pressure on the assessment of AS severity depends primarily on the associated change in mean transvalvular flow rate, rather than on an independent effect of systemic vascular resistance or arterial compliance. This study shed light on afterload dependency AS severity, but still raised another concern that "How about pure effects of transient afterload elevation without increase in heart rate?". This question is worthwhile to be answered because elevated systolic blood pressure in elderly population is usually not from increased sympathetic tone but from increased arterial stiffness caused by large artery fibrosis and calcification, therefore their blood pressure elevation usually is not accompanied by increased heart rate.[5] Current paper by Chang et al.[6] tried to answer this question by using pneumatic compression of both legs, which ideally elevate afterload by increasing systemic vascular resistance and systolic blood pressure without impact on heart rate. They nicely showed effective orifice area of aortic valve was not significantly changed solely by transient elevation of afterload, therefore echocardiographic grading of AS severity would not be significantly affected by transient afterload change. This is good news for physicians as we don't need to consider every hemodynamic status and don't need to seek any correction factors for blood pressure at every image acquisition. However, the both studies conducted by Little et al.[4] and Chang et al.[6] missed some important clinical issues "long-term effects of persistently increased afterload" or "ventricular-afterload mismatch". Transient elevation of blood pressure by artificially modifying vascular tone is not usual clinical scenario, furthermore pneumatic compression also increases preload by augmenting venous return as suggested by increased in LV end-diastolic dimension and early mitral inflow velocity.[6] Preload augmentation accompanied by increased contractility, which is not shown in this study, might compensate effects of after-

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load elevation.[7] Real hidden flaws are in the LV remodeling, preload and contractile reserve. Thickened myocardium associated with small LV cavity, decreased LV filling and impaired midwall fractional shortening altogether contribute to making low stroke volume associated with chronically elevated afterload. Then subclinical afterload mismatch makes worse prognosis,[3] therefore, current issue about afterload problems should not be just focused on transiently elevated systolic blood pressure. We should not forget the real flaw is in the left ventricle. Incorporation of diastolic function, LV mass to volume ratio, midwall fractional shortening[8] or velocity of circumferential myocardial fiber shortening possibly measured by speckle tracking echocardiography[9] would be helpful in further stratifying the risk of AS patients with systemic hypertension.

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