Echocardiographic evaluation of cardiac function response to removal of aortic stenosis: Surgical and trans-catheter aortic valve implantation (TAVI)

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Introduction
Aortic stenosis (AS) is the commonest valve disease in the West, with a prevalence varying between 0.02% in adults under 44 years and 3-9% in those over 80 years of age 1, 2. The disease may remain “silent” and hence unnoticed for years, particularly in the elderly with naturally limited exercise. With the development of symptoms, patients may carry a mortality of 36-52%, 52-80% and 80-90% at 3, 5 and 10 years, respectively if left untreated, with a potential high risk of sudden death 3. Surgical aortic valve replacement (SAVR) used to be the only effective treatment for severe AS, being the second indication of surgery, late regression is slow and might not become complete within the first 6 months. LVH regression predominantly occurs within the first 6 months of surgery, late regression is slow and might not become significant on long-term. Studies reported controversial results regarding the effect of age and gender on LV hypertrophy regression 4, however, a systematic review has shown no statistical association between age and sex with the rate of LV mass regression and change in EF 8.

1. Cardiac function after SAVR

1.1 LV function

Left ventricular hypertrophy (LVH) regression: Aortic valve replacement decreases the LV afterload, resulting in regression of cavity hypertrophy. LV mass regression predominantly occurs within the first 6 months of surgery, late regression is slow and might not become significant on long-term. Studies reported controversial results regarding the effect of age and gender on LV hypertrophy regression 4, however, a systematic review has shown no statistical association between age and sex with the rate of LV mass regression and change in EF 8.

LV function: LV ejection fraction (LVEF) may remain normal in more than 90% of AS patients. However, the long-axis systolic function has been shown to be significantly reduced in AS patients with normal LVEF as assessed by M-mode and Pulsed tissue Doppler velocities 9, 10. LV early diastolic filling and wall thinning rates are also significantly depressed in AS patients, despite being negatively correlated with age, LV peak systolic pressure and wall thickness 11. During the cardiac cycle, the myocardial fibre function changes are not only in the longitudinal direction, but also in the radial and circumferential direction. Analysis of the changes in these three directions, in AS, gives profound information on LV function performance. The new echocardiographic techniques e.g. tissue Doppler Imaging (TDI) and speckle tracking echocardiography (STE) provide accurate evaluation of LV global and segmental myocardial motion (velocity and displacement) as well as deformation (strain and strain rate), respectively. In AS patients, the longitudinal velocity, strain and strain rate (SR) are significantly decreased even in mild AS and deteriorate further as it becomes severe, similar to the previously reported M-mode and Pulsed TDI findings. The reduction in long-axis function is related to the extent of LV hypertrophy 12, 13 and the severity of AS 3, 14. The radial and circumferential strain and SR changes usually occur later than the longitudinal function, which may remain normal in mild AS, but decrease in moderate and severe AS 15. This has been shown by Delgado et al who demonstrated significantly decreased radial and circumferential strain and SR in severe AS with preserved LVEF 16. While another study showed supernormal circumferential strain in patients with normal EF and decreased function when LV systolic dysfunction is present, suggesting that the high circumferential strain may serve as an initial compensatory mechanism for maintaining normal LVEF 17. The differential changes in longitudinal, radial and circumferential fibers reflect the myocardial dysfunction starting at the subendocardium in mild AS and progressing to mid-wall and eventually transmural impairment in severe AS.

After SAVR and immediate drop of LV overload rapid increase of LV myocardial velocities, strain and SR in all three directions occur, as early as 1 week of surgery before global systolic function and LV mass regression 18-20. These improvements may be due to the increased coronary reserve secondary to increased valve effective orifice area, resulting in a more effective myocardial blood supply 21. During mid-term and long-term follow-up, the strain and SR in all directions increase gradually and eventually normalize 16, 21, 22. Such functional recovery in radial and circumferential directions is usually earlier than that in the longitudinal direction. However, even with full LV function recovery at rest, the exercise capacity of these patients remain limited after AVR 23.

LV twist function: The heart normally rotates along its long-axis forming a wringing (twisting) motion during the cardiac cycle. Looking from the apex, the LV base rotates clockwise and the apex rotates counter-clockwise, resulting in a wringing motion. The net rotation difference between apex and base is called twist. During isovolumic relaxation, the rapid untwisting occurs before cavity filling. Studies using magnetic resonance imaging...
imaging (MRI) in AS with preserved LVEF demonstrated an increased apical rotation and delayed LV twist and untwist compared to normals 24,25. Furthermore, the increased apical rotation and LV twist correlate with the severity of AS 26. These changes are considered as compensatory mechanisms for the increased intracavitary pressure and the subendocardial ischemia 25. Another explanation is the potentially rearranged fiber architecture in AS which may alter the torsional deformation of the heart 25. LV twist has also been shown to increase significantly in women with congenital AS and further increase during pregnancy 27 confirming its sensitivity to loading conditions 18. However, this compensatory mechanism is lost in patients with severe LV dysfunction 17. After surgery, the twist function may normalize 26.

1.2 RV function

In AS, the RV function is already abnormal before surgery, even in the absence of secondary pulmonary hypertension. The RV cavity is enlarged, the overall function is reduced although the intrinsic RV myocardial function (measured by SR) remains preserved 28. Despite successful AVR, the preoperative RV dysfunction remains unchanged. These findings suggest an organic remodeling of the RV before surgery that persists afterwards. Furthermore, AVR is known to affect RV function with reduced amplitude of the free wall motion, clinically described as RV long axis function or tricuspid annulus peak systolic excursion (TAPSE), the exact explanation of such disturbance has not been successfully established. Some suggestions for potential mechanisms have been reported including right atrial cannulation, suboptimal RV myocardial preservation or sternal opening 30,31. However, none of these hypotheses has yet proved a satisfactory explanation. A recent study showed that the reduced TAPSE is related to the reversed septal motion after AVR 26,32. With the reversed septal motion after AVR, the time to peak septal displacement become delayed, in contrast to the LV lateral wall which is early and its amplitude exaggerated 26, suggesting a cross talk between LV and RV that allows maintaining global function.

1.3 LA function

One of the hemodynamic consequences of AS is pressure overload which leads to LA dilatation. In AS, the LA peak systolic (reservoir function), early diastolic (conduit function), and late diastolic (active function) longitudinal strain rate are all reduced as well as the LA longitudinal strain [34]. LA late diastolic SR has been shown to be independently related to aortic valve area. After SAVR, the global peak atrial longitudinal strain increases related to trans-aortic mean gradient and aortic valve area changes [35].

2. Cardiac function after TAVI

2.1 LV function

TAVI has proved very useful in alleviating symptoms and improving survival of AS patients. In addition, the procedure has significant effect on LV function. The LV function changes after TAVI are of the same nature as those after SAVR, LHV significantly regresses although not complete after 1 year follow-up 36. Although together with the significant drop of trans-valvular gradient, early improvement of EF and posterior wall myocardial thickening and thinning velocities occur, recovery of strain and SR have been reported as early as 24 hours of the procedure 37. Eight weeks after procedure, TAVI resulted in similar increase of LV lateral wall longitudinal amplitude and myocardial velocity compared to SAVR 38. Meanwhile, similar to SAVR, the decreased longitudinal systolic strain and SR in severe AS increase after 1 month of TAVI, irrespective of changes in EF. The unchanged radial and circumferential strain and SR reported after procedure may be due to the near-normal values before TAVI 39. It seems that the changes of LV myocardial motion and deformation after TAVI or SAVR are more dependent on the baseline patients’ characteristics than the procedure itself since they both decrease the afterload immediately. So far, the effect of TAVI on LV twist function remains to be thoroughly studied.

2.2 RV function

In contrast to SAVR, TAVI has proved to have no negative effect on RV function with TAPSE remaining well preserved and RV free wall systolic velocity increased compared with gender, age and LV function matched SAVR patients after 8 weeks of procedure 32,38.

2.3 LA function

Early after TAVI, the LA reservoir and conduit function improve but LA systolic function remain unchanged, indicating early partial recovery of LA function 40. The LA systolic function recovery may need longer time.

Conclusion

In severe aortic stenosis and normal EF, the three cardiac chambers, LV, RV and LA are disturbed and respond differently to removal of afterload. The LV longitudinal, radial and circumferential strain and SR are reduced and twist is increased, all recover after SAVR and TAVI. In AS, RV cavity is enlarged and its function compromised, but these abnormalities remain up to 6 months of SAVR, which further reduces TAPSE in contrast to TAVI which does not. The LA strain and SR are reduced in AS patients, and improve after successful SAVR and TAVI.

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References


