Longitudinal myocardial shortening in aortic stenosis: ready for prime time after 30 years of research?

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Longitudinal myocardial shortening in aortic stenosis: ready for prime time after 30 years of research?

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Aortic valve replacement (AVR) is recommended for patients having severe aortic stenosis (AS) associated with symptoms and/or left ventricular systolic dysfunction defined as left ventricular ejection fraction (LVEF) of less than 50%. Left ventricular dysfunction may, however, develop insidiously in the asymptomatic patient with severe AS and may eventually become irreversible. Accordingly, some recent studies support the realisation of elective surgery in asymptomatic patients with severe AS to improve their long-term survival.[1] On the other hand, the risk of operative death associated with elective AVR may outweigh the low risk of sudden death that has been reported in asymptomatic patients. The challenge for the clinician is thus to detect left ventricular contractile dysfunction at an early or subclinical stage so that closer follow-up can be instituted or surgical correction performed to prevent the development of irreversible left ventricular dysfunction and adverse outcomes.

In this issue of *Heart*, Cramariuc *et al*[2] report the results of an elegant study in which they examined the relationship between myocardial deformation assessed by speckle tracking imaging and left ventricular geometry in patients with AS (***see page 106***). The main findings of the study are: (1) left ventricular longitudinal myocardial strain is reduced in patients with left ventricular concentric hypertrophy; (2) the extent of longitudinal strain impairment is related to larger left ventricular mass index, higher relative wall thickness ratio (ie, higher degree of concentric remodelling) and more severe stenosis severity.

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Thirty years ago in 1979, Dumesnil and colleagues[3] were the first to report that patients with AS often have selective decreases in left ventricular systolic longitudinal shortening and wall thickening while LVEF remains normal. They explained this discrepancy by the fact that LVEF is influenced by both intrinsic myocardial function and left ventricular cavity geometry (fig 1). There is indeed an independent relationship between LVEF and the relative wall thickness ratio[3 4] and thus, for a similar extent of intrinsic myocardial shortening, the LVEF or any parameter based solely on endocardial displacement (eg, fractional shortening) tends to increase in relation to the extent of left ventricular concentric remodelling (fig 1). Therefore, the increase in wall thickness associated with left ventricular concentric hypertrophy results in a greater contribution of wall thickening to endocardial inward displacement. As a consequence, LVEF remains normal or may even be supranormal in the presence of concentric hypertrophy, despite a decrease in longitudinal shortening.[4] This concept is well illustrated by the results reported by Cramariuc *et al*[2] whereby the LVEF was similar among the different patterns of left ventricular geometry, although left ventricular longitudinal myocardial strain was substantially reduced in patients with concentric hypertrophy.[2] Along with this concept, Poulsen *et al*[5] observed that the regression of left ventricular concentric hypertrophy following AVR results in an improvement in longitudinal myocardial strain and plasma levels of brain natriuretic peptide, whereas LVEF remains unchanged. Moreover, we previously reported that AVR with a stentless bioprosthesis is associated with a lesser residual transvalvular gradient and better recovery of left ventricular longitudinal shortening compared with AVR with a stented bioprosthesis.[6]

From a pathophysiological standpoint, these observations are consistent with the concept that: (1) in pressure overload cardiomyopathy, the increase in wall stress and intramyocardial pressure as well as the reduction in myocardial blood flow occurs mainly in the subendocardium and (2) the subendocardial myocardial fibres are oriented longitudinally. Therefore, the selective impairment in longitudinal myocardial kinetics often observed in AS might be related to the increase in subendocardial wall stress and associated subendocardial ischaemia and fibrosis. These findings also emphasise that parameters of left ventricular systolic function based on endocardial displacement can remain normal despite significant myocardial damage in the subendocardium. In this regard, it is important to emphasise that LVEF is the only index that is included in the guidelines to identify left ventricular systolic dysfunction, which is a class I indication for AVR. Unfortunately, as further illustrated in the present study,[2] LVEF markedly underestimates the extent of myocardial systolic impairment in the presence of left ventricular concentric hypertrophy such as is often the case in AS patients. As a matter of fact, Cramariuc *et al*[7] recently reported that one third of asymptomatic patients with preserved LVEF enrolled in the SEAS trial had a significant impairment of myocardial systolic function. Therefore, a LVEF greater than 50% as well as the absence of symptoms cannot exclude the presence of intrinsic myocardial dysfunction.

In light of the data published in the literature, the analysis of myocardial kinetics in the longitudinal direction provides the most powerful approach to unmask the subclinical myocardial dysfunction that is often not detected by LVEF.[2–5 8 9] Furthermore, the indices of longitudinal displacement and strain are superior to other indices of left ventricular systolic function in predicting symptoms, exercise tolerance and outcomes in AS patients.[8 10 11]

In earlier studies, the left ventricular longitudinal shortening was derived from M-mode or two-dimensional echocardiographic measurements. In subsequent studies, other investigators used peak systolic mitral annulus velocities measured by Doppler tissue imaging because this method was simpler and required fewer measures. In the recent era, longitudinal myocardial strain measured by speckle tracking has emerged as the most promising alternative to detect and quantify intrinsic myocardial systolic dysfunction in AS patients[2 5 9] This method may

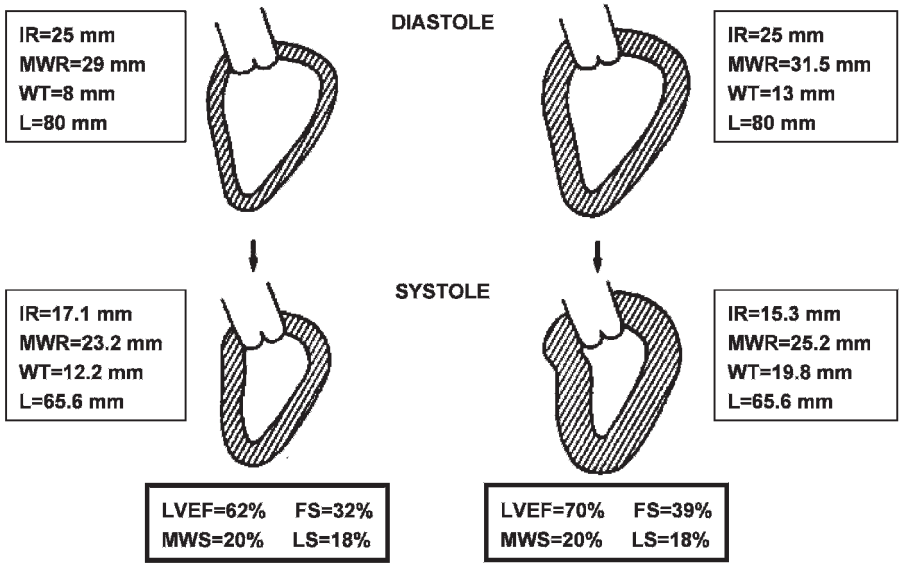

Figure 1 Relationship between left ventricular geometry and myocardial function. This figure shows the schematic representation of changes in left ventricular dimensions in two ventricles with different left ventricular geometries (left: normal; right: concentric remodelling; relative wall thickness (WT) 0.32 vs 0.52) and identical intrinsic myocardial shortening as exemplified by similar values for mid-wall radius shortening (MWS 20%), longitudinal axis shortening (LS 18%) and wall thickening (52%). Due to the effect of geometry, ejection fraction is higher in the ventricle with concentric remodelling (left ventricular ejection fraction (LVEF) 70% vs 62%) because of a greater net gain in wall thickness during systole (7 mm vs 4 mm) and thus greater relative contribution of thickening to ejection (32% vs 23%). Therefore, if intrinsic longitudinal shortening were to be decreased as in aortic stenosis, the LVEF and internal radius shortening (ie, fractional shortening; FS) could still remain normal due to the compensatory effect of geometry. Adapted with permission from Dumesnil and Shoucri.[4] IR, internal radius; L, length of the ventricle; MWR, mid-wall radius.

be superior to other modalities because it is angle independent and thus potentially more reproducible. Stress corrected mid-wall shortening has also been shown to be useful to unmask intrinsic myocardial dysfunction in patients with AS and/or arterial hypertension[7] and, in the present study,[2] this index revealed important differences in myocardial systolic function among the four patterns of left ventricular geometry, whereas LVEF did not differ between these patterns. This index, however, has the same limitation of the M-mode or two-dimensionally derived indices of longitudinal shortening in the sense that its calculation is more complex and requires several measures, which may increase the risk of measurement error.

The results presented in this study[2] further confirm that the geometry of the ventricle is a strong determinant of myocardial systolic function, and left ventricular concentric hypertrophy is associated with a worst degree of myocardial impairment. Therefore, the assessment of left ventricular geometry and function in patients with AS should be more comprehensive and go beyond the sole measurement of left ventricular mass and ejection fraction. This evaluation should also include relative wall thickness to assess the degree of concentric remodelling and longitudinal myocardial strain to identify and quantify myocardial systolic dysfunction properly. Given that these indices can now be measured routinely and reproducibly, we thus believe that they should be incorporated into the routine echocardiographic follow-up of patients with AS.

Competing interests: None.

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