Emerging role of echocardiographic strain/strain rate imaging and twist in systolic function evaluation and operative procedure in patients with aortic stenosis

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Summary

Systolic function of the left ventricle is vital for patients with aortic stenosis. Unfortunately, the most widely used clinical parameter, the left ventricular ejection fraction, is not sensitive enough, especially for patients with left ventricular hypertrophy. Echocardiographic strain/strain rate and twist are emerging parameters for left ventricular systolic and diastolic function evaluation. Aortic stenosis could reduce strain/strain rate while magnifying twist. Furthermore, strain/strain rate correlates well with the prognosis of patients with aortic stenosis. Most importantly the circumferential strain, strain rate and twist also play a role in differentiating cardiac compensation or decompensation. In any case, these parameters could normalize after successful surgical aortic valve replacement or transcatheter aortic valve replacement. Regardless of these advantages, clinical evidence is needed to ensure their usefulness.

Keywords: Aortic stenosis • Strain • Strain rate • Twist • Cardiac systolic function

INTRODUCTION

Aortic stenosis (AS) is one of the common valvular heart diseases, mostly caused by rheumatic fever, valvular degeneration, antiphospholipid syndrome, congenital aortic bicuspid valve and so on. In general, aortic valve replacement (AVR) or valvuloplasty is beneficial for symptomatic patients to improve their haemodynamic and cardiac function. However, quite a few patients do not experience cardiac function improvement or even deterioration after successful aortic valve surgery [1]. Therefore, cardiac surgeons are somewhat confused with regards to how to select appropriate patients for an aortic valve procedure as well as how to judge the prognosis. Selection criteria for AVR or valvuloplasty includes symptoms, left ventricular ejection fraction (LVEF), size of left ventricle (LV), atrial fibrillation and other indexes. LVEF is the most important parameter, but until now conservative echocardiography could not fully ensure its reliability [2], especially in patients with abnormal haemodynamics, left ventricular hypertrophy or ventricular dilatation.

Echocardiographic strain/strain rate (SR) and torsion are emerging parameters on myocardial deformation. In this paper, we reviewed and emphasized the role of these useful parameters on left ventricular systolic function evaluation, prognosis and operative procedures. A complete literature search on EMBASE, PubMed, MD Consult and ISI Web of Science from 1998 to 2012 was conducted. We screened the abstracts and listed out related studies. All the strain/SR and twist-related studies on AS patients cited in this paper are full-text papers.

ECHOCARDIOGRAPHIC STRAIN/STRAIN RATE IMAGING AND TORSION

The double-helical structure of LV myofibres shortens in the longitudinal and circumferential directions and thickens in the radial direction at systole. Strain and SR are regarded as objective parameters of directional deformation and can detect subclinical impairment. Strain is an index that refers to the amount of myocardial deformation in one direction normalized to its initial dimension, written as:

$$S = \frac{L - L_0}{L_0}$$

where $L$ is the length after deformation and $L_0$ is the length before deformation. It can be applied to evaluate the systolic function at longitudinal, circumferential and radial directions (Fig. 1). SR is the rate of deformation within a time unit (1 s), calculated as:

$$\text{SR} = \frac{V_1 - V_2}{\Delta L}$$
where $V_1$ and $V_2$ are velocities at two points on ventricular muscles, and $\Delta L$ is the distance between these two points. It has been demonstrated that strain is a non-dimensional quantity which is independent of wall thickness and motion. This means that myocardial strain will not be affected by cardiac motion, rotation or the function of adjacent segments [3, 4].

In addition to local myocardial motion, torsion is a rotatory measurement for the LV in short-axis views. Looking from the apex, counterclockwise rotation is defined as positive and clockwise as negative. Twist is sometimes used simply to mean wringing, while torsion means the basal-to-apex gradient in the rotation angle (Fig. 2) [5]. Apical rotation and twist mostly represent synchronous changes, which increase in AS and decrease in myocardial infarction, dilated cardiomyopathy and heart failure [6].

To quantify myocardial strain, SR and twist, there are three available echocardiographic imaging modalities: tissue Doppler imaging (TDI), two-dimensional speckle-tracking imaging (2D-STI) and three-dimensional speckle-tracking imaging (3D-STI). By means of these imaging techniques, information on myocardial strain, SR and torsion can be derived (Fig. 3).

**Tissue Doppler imaging**

TDI is a Doppler technique that allows quantification of myocardial tissue velocities. By using a high-pass filter (>100 Hz), the high amplitude but low-velocity myocardial velocities can be measured. Through spatial derivation of the derived velocity, the strain and SR can be obtained [7].

**Two-dimensional speckle tracking**

2D-STI allows frame-by-frame tracking of natural acoustic markers within the myocardium in standard echocardiographic images. Unlike TDI, 2D-STI is not a Doppler-based technique and allows direct derivation of myocardial strain that is angle independent. 2D-STI-derived SR is obtained through temporal derivation of the 2D strain data, and spatial integration of 2D SR results in 2D myocardial velocity [7].

**Three-dimensional speckle tracking**

With regard to the 3D technique, the speckles are tracked inside the three-dimensional scan volume, and irrespective of their direction, myocardial motion and deformation of the entire LV can be assessed in all three spatial dimensions through the entire cardiac cycle [7].

The reproducibility of TDI and 2D-STI is acceptable. However, when radial deformation was measured with TDI, both intra-observer variability (<8%) and interobserver variability (<16%) were significantly higher [8, 9]. Meanwhile, the intra- and inter-observer agreement for strain measurement in 3D-STI were more encouraging (both <3%) [10].
As several studies identified, myocardial deformation was impaired due to AS, and the deterioration was related to the severity of AS [11–13]. Ng et al. [12] have found that there is a progressive stepwise impairment in longitudinal/radial/circumferential strain and SR along with the severity of AS progression (all \( P < 0.001 \)). Miyazaki et al. [14] also suggested that global longitudinal strain manifested significant differences among mild/moderate/severe AS groups (\( P = 0.003 \)). A similar outcome was also reported by Delgado et al. [11], showing that multidirectional deformations decreased except radial strain. Moreover, Marechaux et al. [13] conducted research to assess the relationship between valvulo-arterial impedance and global longitudinal strain, and a negative correlation was certified (\( r = -0.41, P < 0.0001 \)).

Stress testing (low-dose dobutamine or exercise stress echocardiography) is recommended for asymptomatic patients with severe AS, especially for differentiating true or pseudo-severe AS and detecting contractile reserve (compensation) [15]. Global 2D longitudinal strain as determined by the speckle-tracking method has the potential to provide more precise information on LV function and contractile reserve during stress testing [16]. Donal et al. [17] verified the decrease of longitudinal strain be it at rest or during exercise stress in AS patients. Furthermore, changes in longitudinal strain during exercise in AS patients was also lower than in the normal control. Donal et al. [17] and Lafitte et al. [18] both demonstrated that lower longitudinal strain was detected in AS patients with abnormal exercise testing scores compared with AS patients with normal exercise testing scores.

Furthermore, the elevated LV afterload can induce alteration of torsion. Compared with normal subjects, patients with AS manifested increased apical rotation (13.0 ± 5.8° vs 7.6 ± 2.6°, \( P < 0.001 \)) and twist (19.7 ± 5.7° vs 12.9 ± 3.2°, \( P < 0.001 \)), while basal rotation remained normal [19]. Similar outcomes were obtained by van Dalen et al. [20] and Popescu et al. [21]. They proved that apical rotation (\( R^2 = 0.34, P < 0.001 \)) and twist (\( R^2 = 0.30, P < 0.001 \)) correlated well with aortic valve area indexed by body surface area. In addition, pregnancy, which induces volume overload, generated augmented twist, and in gravidas with AS, twist was prone to a stepwise increase compared with the baseline while longitudinal strain showed little changes [22]. Tammo et al. also demonstrated the elevation of the torsion-to-shortening ratio in AS patients, which was considered as a more sensitive parameter of subendocardial ischaemia [23]. Additionally, AS could induce an increase in the peak apical de-rotation rate and time-to-peak diastolic de-rotation rate [20, 21]. Nevertheless, Laser et al. [24] did not think there was a relationship between the severity of disease and twist.

**CIRCUMFERENTIAL STRAIN AND TWIST ARE VALUABLE IN THE IDENTIFICATION OF LEFT VENTRICLE COMPENSATION AND DECOMPENSATION**

Cardiac compensation occurs due to altered protein composition and stimulation in the cells and extracellular matrix, which
initially manifests in enhancement of the contractile force and hypertrophy without dilatation. Normal cardiac output is maintained during compensation. But as the impairment persists, cardiac output decreases and cannot afford normal consumption, resulting in symptoms and LV decompensation. LV hypertrophy is a state of pressure-overloaded remodelling, which is characterized by histological fibrosis and myocyte degeneration [25]. It is a deleterious pathology despite being partially reversible after pressure unloading [26]. It is difficult to predict reversibility after AVR for patients with AS. Normal LV deformation could be considered as the compensation which foreshows a fine response to AVR, whereas low deformation could predict a relative high possibility of cardiac events [27]. Carasso et al. [28] performed research on responses of deformation to afterload elevation on patients with severe AS. Accidentally, impact changes of longitudinal and circumferential strain were detected. Thus, it was hypothesized that circumferential strain may increase with myocardial compensation and decrease with decompensation. This hypothesis was verified by the following study. Carasso et al. [29] clearly demonstrated the differential mechanics between compensation and decompen-sation of AS using strain imaging. Compensatory LV showed an increased apical rotation angle in patients with 50% > LVEF > 35% and high circumferential strain in patients with preserved LVEF, whereas dec-ompensation showed decreased circumferential strain and an apical rotation angle.

Circumferential strain, apical rotation and twist have differential changes on occasions of compensation and decompen-sation. In other words, circumferential strain and apical rotation or twist may be parameters for differentiating compensation or decompensation for AS patients. Elevated apical rotation and twist were also predicted to be useful indexes indicating compensation by Lindqvist et al. [19] and van Dalen et al. [20].

**ALTERATIONS OF STRAIN OR STRAIN RATE POST-AORTIC VALVE REPLACEMENT**

As listed in Tables 1 and 2, myocardial deformation improved progressively after successful AVR [11, 28, 30]. Although Rost et al. [31] indicated that there was no significant improvement in strain observed 1 week after AVR along with unchanged LVEF, longer follow-up still showed increased deformation at 6 months after AVR. Meanwhile, Hyodo et al. revealed that the ratio of sub-endocardial to subepicardial radial strain could significantly differ-entiate the severity of AS, which improved as early as 7 days after AVR while subepicardial radial strain showed little changes [32]. Notably, to ascertain whether amelioration of longitudinal deformation presents at 1 week post-AVR, more research studies are needed. Carasso et al. [28] discovered that longitudinal systolic strain increased from $-12.8 \pm 1.7\%$ (lower than normal) to $-15.9 \pm 2.2\%$, whereas mid-LV circumferential strain decreased from $-27.0 \pm 5.1\%$ (higher than normal) to $-22.3 \pm 4.9\%$ at early follow-up post-AVR (7 ± 3 days) in AS patients with preserved LVEF. But Carasso et al. [29] carried out further research on AS patients with EF < 50% and found that circumferential strain decreased before AVR compared with normal controls, and acquired significant improvement after AVR. Twist and apical rotation increased in compensated patients and significantly normalized after AVR [11], but normalization of twist in decom-pensated patients after AVR was uncertain [29]. Otherwise, chil-dren with coarctation or valvular AS acquired decreased stress gradient and twist ($11.8 \pm 4.9^\circ$ vs $16.7 \pm 6.7^\circ$, $P < 0.001$) due to interventional catheterization [24].

As such, we can say that longitudinal and radial strain/SR, as well as twist, decrease before AVR and partially recover due to surgical procedure. Alterations of circumferential strain/SR are reflected on the status of compensation or decompensation. Anyway, AVR always induces normalization of all directional deformations.

**CHANGES OF STRAIN/STRAIN RATE AFTER TRANSCATHETER AORTIC VALVE IMPLANTATION**

Transcatheter aortic valve implantation (TAVI) is an emerging alternative technique to surgical AVR, which is especially suitable for high-surgical-risk patients with severe symptomatic AS, such as senile patients and those with severe heart failure, coagulation disorder and chronic kidney injury [33]. As reported, the survival rate of high-risk patients who underwent TAVI was 92.9% at 30 days after the procedure, 78.6% at 1 year and 73.7% at 2 years [34]. Table 3 is a summary on the alteration of deformation post-TAVI detected by strain and strain rate. Bauer et al. [35] have demonstrated that TAVI improved longitudinal deformation in patients with AS just 24 h after the

<table>
<thead>
<tr>
<th>Study</th>
<th>Method</th>
<th>Machine</th>
<th>Operation</th>
<th>Follow-up</th>
<th>Parameters detected before operation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iwahashi et al. [30]</td>
<td>21s-AS</td>
<td>DTI, PC3.0, GE</td>
<td>AVR</td>
<td>2 w</td>
<td>Longitudinal S/SR of 18 segments</td>
</tr>
<tr>
<td>Rost et al. [31]</td>
<td>40s-AS</td>
<td>STI, Vivid-7, GE</td>
<td>AVR</td>
<td>1 w, 6 m</td>
<td>Longitudinal/radial S in 18 segments; circumferential peak S in 6 segments</td>
</tr>
<tr>
<td>Carasso et al. [28]</td>
<td>45AS</td>
<td>DTI, VVI, Siemens</td>
<td>AVR</td>
<td>1 w, 5 m</td>
<td>Longitudinal S/systolic and early diastolic SR of 18 segments</td>
</tr>
<tr>
<td>Lindqvist et al. [19]</td>
<td>28s-AS</td>
<td>STI, version 8, GE</td>
<td>AVR</td>
<td>6 m</td>
<td>Circumferential S/systolic SR of mid-LV; Apical and basal rotation; twist</td>
</tr>
</tbody>
</table>

AS: aortic stenosis; s-AS: severe aortic stenosis; STI: speckle-tracking imaging; DTI: Doppler tissue imaging; AVR: aortic valve replacement; m: month; w: week; LV: left ventricle.
### Table 2: Alterations of strain/SR post-AVR

<table>
<thead>
<tr>
<th>Study</th>
<th>Normal</th>
<th>Pre-AVR</th>
<th>1 week</th>
<th>2 weeks</th>
<th>6 months</th>
<th>17 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Longitudinal S (%)</td>
<td>2.3 ± 1.6 (P &lt; 0.0001)</td>
<td>1.1 ± 1.6 (P &lt; 0.0001)</td>
<td>2.5 ± 1.6 (P &lt; 0.0001)</td>
<td>3.4 ± 1.6 (P &lt; 0.0001)</td>
<td>3.6 ± 1.6 (P &lt; 0.0001)</td>
<td>3.8 ± 1.6 (P &lt; 0.0001)</td>
</tr>
<tr>
<td>Longitudinal SR (s⁻¹)</td>
<td>2.4 ± 0.3 (P &lt; 0.0001)</td>
<td>1.3 ± 0.3 (P &lt; 0.0001)</td>
<td>2.5 ± 0.3 (P &lt; 0.0001)</td>
<td>3.4 ± 0.3 (P &lt; 0.0001)</td>
<td>3.6 ± 0.3 (P &lt; 0.0001)</td>
<td>3.8 ± 0.3 (P &lt; 0.0001)</td>
</tr>
<tr>
<td>Radial SR (s⁻¹)</td>
<td>2.2 ± 0.3 (P &lt; 0.0001)</td>
<td>1.3 ± 0.3 (P &lt; 0.0001)</td>
<td>2.5 ± 0.3 (P &lt; 0.0001)</td>
<td>3.4 ± 0.3 (P &lt; 0.0001)</td>
<td>3.6 ± 0.3 (P &lt; 0.0001)</td>
<td>3.8 ± 0.3 (P &lt; 0.0001)</td>
</tr>
<tr>
<td>Circumferential SR (s⁻¹)</td>
<td>2.0 ± 0.3 (P &lt; 0.0001)</td>
<td>1.2 ± 0.3 (P &lt; 0.0001)</td>
<td>2.4 ± 0.3 (P &lt; 0.0001)</td>
<td>3.3 ± 0.3 (P &lt; 0.0001)</td>
<td>3.5 ± 0.3 (P &lt; 0.0001)</td>
<td>3.7 ± 0.3 (P &lt; 0.0001)</td>
</tr>
<tr>
<td>Apical rotation (°)</td>
<td>19.1 ± 4.5 (P &lt; 0.0001)</td>
<td>11.3 ± 4.5 (P &lt; 0.0001)</td>
<td>19.5 ± 4.5 (P &lt; 0.0001)</td>
<td>21.4 ± 4.5 (P &lt; 0.0001)</td>
<td>23.3 ± 4.5 (P &lt; 0.0001)</td>
<td>25.2 ± 4.5 (P &lt; 0.0001)</td>
</tr>
<tr>
<td>Twist (%)</td>
<td>12.9 ± 2.3 (P &lt; 0.0001)</td>
<td>7.6 ± 2.3 (P &lt; 0.0001)</td>
<td>12.9 ± 2.3 (P &lt; 0.0001)</td>
<td>19.1 ± 2.3 (P &lt; 0.0001)</td>
<td>23.3 ± 2.3 (P &lt; 0.0001)</td>
<td>25.2 ± 2.3 (P &lt; 0.0001)</td>
</tr>
</tbody>
</table>

### STRAIN/STRAIN RATES ARE PROMISING PARAMETERS INDICATING THE PROGNOSIS AFTER AVR

Bauer et al. [27] suggested that systolic radial strain/SR of the posterior wall predicted good or bad responses after AVR on patients of AS. Systolic SR and strain of patients obtaining good responses were both higher than those of patients not obtaining a good response (1.8 ± 0.4 s⁻¹ vs 1.4 ± 0.6 s⁻¹, P < 0.02; 38 ± 10% vs 30 ± 15%, P < 0.02). While the cut-off value was applied, patients with a peak strain rate (pSR) > 2 s⁻¹ or peak radial strain > 36.2% could acquire more improvement of their 6-min walk distance at the 3-month follow-up and fewer recurrence of heart failure than patients with pSR < 2 s⁻¹. The research of Zhao et al. [39] found that oxygen consumptions at peak exercise in patients who underwent surgical AVR were less than that in the healthy group who had the same LVEF, and significantly correlated with systolic global longitudinal SR (r = 0.60, P = 0.0077), which was the only independent predictor in a multivariate model. As we know, natriuretic peptides, a parameter correlated with several cardiovascular events and prognosis [40], was directly correlated with the time to peak untwist rate and the time to peak apical de-rotation rate [21]. The research of Poulsen et al. [41] showed an increment of the mean longitudinal strain from −9 ± 4% to −14 ± 4% at 12-month post-AVR, which was independently correlated with changes of natriuretic peptides (r = −0.67, P < 0.001).

Incidentally, there was a significant relationship between longitudinal strain (R² = 0.6132) or mid-LV circumferential strain (R² = 0.7692) and LVEF, but the vector of mid-LV longitudinal and circumferential strain was better correlated with the LVEF (R² = 0.8037) [29]. Correlations between longitudinal S/SR and LVEF in patients of low-flow low-gradient aortic stenosis (LF-LG AS) also achieved consistency [42]. While LVEF is the most common clinical parameter on surgical risk and prognosis in patients with aortic valve diseases, we can also say that strain and SR could predict patient prognosis.
APPLICATION OF STRAIN AND STRAIN RATE IN LOW-FLOW LOW-GRADIENT AORTIC STENOSIS

LF-LG AS is defined as the combination of an effective orifice area \( \leq 0.6 \text{ cm}^2 \) or \( \leq 0.6 \text{ cm}^2/\text{m}^2 \) when indexed by the body surface area and a low mean transvalvular gradient (<40 mmHg), accompanied with a persevered or low LVEF. Dobutamine stress echocardiography (DSE) is recommended to test the LV flow reserve (increase in stroke volume >20%), which is useful to estimate operative risk but does not predict recovery of LV function, symptomatic improvement and late survival after operation [43]. Recently, Bartko et al. [42] carried out a research on the relationship between longitudinal strain/SR and survival rate of LF-LG AS, and found that these two parameters measured on DSE were the univariate predictors, and that peak longitudinal SR could add incremental prognostic value.

Table 3: Outcomes of related studies on TAVI

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Technique</th>
<th>Machine</th>
<th>Follow-up</th>
<th>Parameters detected before operation</th>
<th>Changes of parameters pre-/post-TAVI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bauer et al. [35]</td>
<td>8</td>
<td>DTI</td>
<td>VingMed</td>
<td>24 h</td>
<td>Anterior wall peak systolic strain</td>
<td>11 ± 9 vs 18 ± 7%, P = 0.02</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Anterior wall peak systolic SR</td>
<td>0.8 ± 0.5 vs 1.5 ± 0.3 s^−1, P = 0.002</td>
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<td></td>
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<td></td>
<td>Posterior wall peak systolic S</td>
<td>11 ± 5 vs 17 ± 9%, P = 0.02</td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>Posterior wall peak systolic SR</td>
<td>1.0 ± 0.3 vs 1.9 ± 0.7 s^−1, P = 0.009</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td>Longitudinal strain</td>
<td>11 ± 3 vs 12 ± 3%, P = 0.64</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Longitudinal strain of patients without new conduction abnormalities (9 cases)</td>
<td>11 ± 3 vs 13 ± 3%, P &lt; 0.05</td>
</tr>
<tr>
<td>Tzikas et al. [37]</td>
<td>27</td>
<td>STI</td>
<td>iE33, Philips</td>
<td>6 d</td>
<td>Longitudinal strain of patients with new conduction abnormalities (18 cases)</td>
<td>11 ± 4 vs 11 ± 2%, P &gt; 0.05</td>
</tr>
<tr>
<td>Grabskaya et al. [38]</td>
<td>36</td>
<td>STI</td>
<td>Vivid7, GE</td>
<td>1 m</td>
<td>Longitudinal strain</td>
<td>-15.8 ± 3.6 vs -17.6 ± 3.1%, P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Longitudinal SR</td>
<td>-1.03 ± 0.21 vs -1.21 ± 0.19 s^−1, P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Circumferential and radial strain/SR</td>
<td>P &gt; 0.05</td>
</tr>
</tbody>
</table>

TAVI: transcatheter aortic valve implantation; DTI: Doppler tissue imaging; STI: speckle-tracking imaging; h: hour; d: day; m: month.

LIMITATION OF STRAIN/STRAIN RATE IMAGING AND TWIST

S/SR and twist have been certified as valuable tools in the understanding and assessment of myocardial motion, function and mechanics. Owing to lack of substantial research, strain/SR and twist have not been utilized in everyday clinical practice and have remained research tools. LVEF is still the most important parameter on left ventricular function and diagnosis.

Moreover, the three imaging techniques have various limitations. Like all Doppler-derived measurements, TDI is limited by angle dependency (the greater the insonation angle, the lower is the measured velocity); this is the bane of worse reproducibility in the radial direction [8]. Owing to the effect of respiration and tethering effects of the surrounding myocardium, the area of interest moves in and out of the scanning beam during the cardiac cycle and this makes tracking difficult. Furthermore, TDI is susceptible to noise arising from the blood pool [3, 7]. Although there is no angle limitation, 2D-STI is prone to a higher degree of error while motion is perpendicular to the ultrasound beam. Similar to TDI, speckle patterns are not constant in serial frames [44]. 3D-STI has emerged as a further advancement to provide greater insight, but is much less popular than TDI or 2D-STI due to the limited availability of echo systems. In general, 2D-STI is currently used most widely in clinical practice.

CONCLUSION

Systolic strain/SR and twist are emerging parameters developed during this decade. Their challenging roles in coronary artery disease, ventricular disynchrony, chronic heart failure and right ventricular function have been discussed. Their roles in aortic valve disease and surgery (AVR or aortic valve reconstruction or TAVI) also cannot be ignored. These parameters directly delineate the motion of the LV and demonstrate the function of the LV. We express several opinions and desired research directions: (i) strain, SR and torsion, especially longitudinal strain and SR, can reflect the severity of AS and systolic function of the LV; (ii) circumferential strain/SR and twist seem to be promising parameters on differentiating LV compensation or decompensation, which is important for preoperative evaluation. But as the status of compensation or decompensation has no concrete diagnostic criteria, sequential research studies are needed for identification of a cut-off value which could indicate operative mortality and prognosis; however, we can say decreased circumferential strain and SR indicates higher operative risk and a worse prognosis; (iii) deformations of the LV are partly normalized after AVR, be it during compensation or decompensation; however, the length of time needed for deformation recovery is unclear—1 week or 2 weeks? (iv) TAVI is a minimally invasive technique, but whether the occurrence of new conduction abnormalities affects the recovery of LV deformation is still not clear; (v) considering the fuzziness of myocardial imaging and the inaccuracy of ventricular wall recognition, more advanced devices and probes are necessary.

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