Rocking will tell it

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This editorial refers to ‘Toward understanding response to cardiac resynchronization therapy: left ventricular dyssynchrony is only one of multiple mechanisms’1, by C. Parsai et al., on page 940 and ‘Low-dose dobutamine stress echo to quantify the degree of remodelling after cardiac resynchronization therapy’2, by C. Parsai et al., on page 950.

During the past years, cardiac resynchronization therapy (CRT) has become a recognized treatment option for patients with heart failure and conduction delays. In the meantime, the principle of re-synchronizing a dysynchronous ventricle can be regarded as established.1 The challenge of correctly identifying the patient candidates who will benefit from this costly and not completely non-invasive therapy, however, remains.

With its wide availability, feasibility, and its potential to analyse regional myocardial function with excellent temporal and good spatial resolution, echocardiography may be regarded as the ideal imaging modality for CRT patient selection. Many attempts have been made in the past years to utilize tissue Doppler for this task. A plethora of parameters came upon us, mostly based on differences in timing of systolic velocity peaks in up to 12 different myocardial regions.2 Only a few attempts have been made, however, to analyse the underlying mechanics of these measurements and to optimize measurement methods for them.

There are several reasons, why velocity peak-based parameters are suboptimal for analysing left ventricular dyssynchrony. First, there is no direct relationship between regional velocity and regional myocardial contraction.3 This is true in particular in a dysynchronous ventricle, where one region of the ventricular myocardium pulls the other and passive motion is a common finding. Secondly, peak velocities are subject to afterload and their timing does not reflect the timing of mechanical activation. Thirdly, parameters calculated from timing of peak velocities in several segments relinquish precious regional information on the contraction sequence. In addition, measurement reproducibility worsens with the number of measurements needed. As a consequence, the excellent results of smaller, single-centre studies using velocity peak-based parameters4 could not be reproduced in larger trials,5 which put false colours upon the—in principle—convincing concept of measuring what you want to treat, i.e. the mechanical dyssynchrony of the left ventricle (LV). Besides that, dyssynchrony is probably the most important, but not the only factor influencing the success of CRT in an individual patient. Suboptimal lead placement and device settings, limited myocardial viability, and features of the underlying disease may have a confounding impact.

The two recent studies of Parsai et al.6,7 have shed refreshingly new light on the scene. In ‘Toward understanding response to cardiac resynchronization therapy: left ventricular dyssynchrony is only one of multiple mechanisms’,6 the authors try first to separate different pathomechanisms that contribute to the deterioration of LV function in heart failure patients. Not only intraventricular dyssynchrony, but also disturbed atrioventricular coupling and interactions between the right and left ventricle are identified as aetiological factors. The successful treatment of these factors was mostly associated with reverse remodelling or clinical improvement. It may be the subject of discussion as to whether, given its rather low specificity, the suggested clinical path for identifying responders is sufficiently supported by data, but it is certainly to the merit of this study that the authors have refrained from a mono-causal approach to this complex disease.

By far the largest group of the study population presented with intraventricular dyssynchrony. Parsai et al. suggest using a phenomenon for its detection which represents the early septal contraction in an LV with left branch bundle block (LBBB). It is the same early septal contraction that causes the ‘apical rocking’ which can be typically visualized in an echocardiographic four-chamber view of a dysynchronous heart. As a study from our group has shown, this parameter is a surrogate, reflecting both temporal and functional inhomogeneities in the LV with LBBB.8 Parsai et al. call the phenomenon ‘septal flash’ and suggest objectifying it by demonstrating an early septal excursion in a parasternal grey-scale or colour tissue Doppler M-mode. With the ‘septal flash’, Parsai et al. introduce a novel and elegant approach to assess dyssynchrony. It overcomes several of the above-mentioned limitations of peak velocity-based parameters, but avoids the challenges of myocardial deformation measurements. The parameter is

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simple, independent from longitudinal velocity peaks, and retains the important regional information on the myocardial contraction sequence. Certainly, further studies are needed to define cut-off values more clearly and to determine inter- and intra-bserver variability, but the approach appears promising.

With their second article ‘Low-dose dobutamine stress echo to quantify the degree of remodelling after cardiac resynchronization therapy,’ Parsai et al. remain in the same area of interest. They investigate the value of dobutamine stress echocardiography for the identification of potential CRT responders. The concept is striking: similar to akinetic viable myocardium—which despite being functional—is too weak to shorten against the imposed load, the weakness of the septum of a remodelled LV with LBBB may mask its typical early contraction and with this the apical rocking and the ‘septal flash’. Consequently, similar to the viability assessment in ischaemic disease, myocardial contraction—and with this sign of dyssynchrony—may also be provoked by an inotropic stimulus in the case of a dyssynchronous LV. The study proves the concept. From 21 patients who showed no ‘septal flash’ at rest, in five the ‘septal flash’ could be stimulated. All but two patients with ‘septal flash’ became responders. Only one patient with septal flash did not respond. Although not explicitly mentioned in the text, the reported numbers allow calculation of a high sensitivity and specificity for identifying responders of 82 and 88%, respectively, already at rest and an impressive 97 and 88% with low-dose dobutamine. It is interesting that despite comparable study population characteristics, this good performance of the ‘septal flash’ parameter at rest was not fully reached in their multicentre study.6 This underlines the need for a good definition and further investigations on the measurement variability of this promising parameter.

The stress echo study of Parsai et al. also presents remarkable pathophysiological findings. It reveals that increasing dyssynchrony with low-dose dobutamine is associated with response to CRT, indicating that in such patients dyssynchrony is indeed a factor contributing to the functional deterioration of the LV. In contrast, non-responders showed no increasing dyssynchrony and, even more interestingly, showed an improving stroke volume during the stress test.

In summary, the two studies by Parsai et al. are inspiring work, contributing to the understanding of the multifactorial process of functional deterioration in heart failure patients, introducing a new, echocardiographic approach to assess the well known phenomenon of early septal contraction, and showing that this phenomenon, maybe even provoked by an inotropic challenge, is of clinical relevance.

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