Will mechanical dyssynchrony one day impact our management of chronic heart failure patients?

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Mechanical dyssynchrony along with its role in cardiac resynchronization therapy (CRT) has been studied for over 10 years now. Although there are ‘believers’ in imaging techniques and echocardiography specifically, there are no recommendations for their use in clinical practice or guidelines for CRT.

QRS duration remains the gold-standard for selecting patients for CRT and predicting their responses. The key importance of QRS width and morphology, so as to predict responses to CRT, has been demonstrated recently. According to the results of the RethinQ trial involving 172 patients with heart failure and narrow QRS, CRT did not improve peak oxygen consumption or LV remodelling at the 6-month follow-up. It has also been demonstrated that the correlation between mechanical dyssynchrony and QRS width was less than perfect. Thus, despite the 30% rate of non-response to CRT, it is challenging for imaging techniques to demonstrate substantial value, at least as to the therapeutic indication.

Is mechanical dyssynchrony a useless parameter, or do we still not know how to quantify it correctly and with sufficient robustness?

In a prospective, multicentre study on patients with non-ischaemic dilated cardiomyopathy, D’Andrea et al. performed exercise stress echocardiographies with centralized core laboratory analysis. We should emphasize that they used—appropriately—a core laboratory experienced in assessing mechanical dyssynchrony. First, one might be surprised that dyssynchrony was assessed during exercise when this assessment is already so challenging at rest. But, although exercise stress echocardiography has been evaluated only weakly to date, it is probably a very relevant approach for assessing the impact of load changes on mechanical dyssynchrony. Moreover, it is also a physiological approach to analysing the heart globally, without focusing only on dyssynchrony.

D’Andrea et al. assessed mechanical dyssynchrony based on one technique and one echo-system. Their tissue Doppler-based analysis started over 4 years ago. Their results were interesting as they demonstrated that in addition to left ventricular end-diastolic volume, restrictive mitral inflow pattern, and mitral regurgitation severity, the exercise-induced intraventricular dyssynchrony was independently correlated with clinical outcomes (combined endpoint of death, heart transplant, or assist device implantation). Mechanical dyssynchrony can be valuable, but it is important to stress that imaging techniques provide some information that might impact cardiomyopathy assessment: diastolic function, systolic function, right heart function, valvular heart disease, and the degree of fibrosis, in addition to clinical, electrocardiographic, and biological data, might all affect patient management and outcomes.

Is tissue Doppler the best tool for assessing dyssynchrony? RethinQ and Prospect are discouraging studies. Suggesting otherwise, studies, such as MADIT echocardiographic substudy or the TARGET trial, are provided more optimistic observation, considering the speckle tracking assessment of strains for dyssynchrony quantification.

Today, we would probably prefer a mechanical dyssynchrony assessment based on deformation imaging because of this method’s robustness. It is easier to understand from a pathophysiological standpoint than myocardial velocity imaging, which sometimes returns multiple peaks. Nevertheless, we have to keep in mind that, while there are ‘believers’, there is no validated and agreed-upon the gold-standard technique for assessing mechanical dyssynchrony. A multimodality assessment, therefore, remains necessary, although D’Andrea’s et al. results are still very interesting to note. These prognostic data should not be interpreted as encouragement to implant any CRT in patients with narrow QRS, but it encourages continued investigation of mechanical dyssynchrony in patients with narrow and broad QRS, depressed the left ventricular ejection fraction, and preserved the ejection fraction. We very probably have a lot to learn, but if we do find the highly reproducible and feasible tool or tools that we are looking for, then perhaps a therapeutic study based on imaging findings would be arguable.
Even considering the disappointment of some past studies, a strong validation of the value of quantifying mechanical dyssynchrony using imaging techniques is forthcoming. In addition to dyssynchrony, several components of heart function can be quantified. Multiparametric approaches integrated into scores (based on multivariate analyses like the one performed by D’Andrea et al.) might be useful for implementing dyssynchrony, like other parameters, into clinical practice.

We will make progress in the assessment of dyssynchrony by increasing its feasibility and robustness. Standardization of this assessment will then be possible, allowing imaging techniques to be used for new therapeutic targets and also, hopefully, along with the ECG, for predicting non-response to CRT.

References
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