How should tissue Doppler tracings be measured?

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Tissue Doppler, an echocardiographic modality measuring velocities of solid cardiac structures by pulsed-wave or colour Doppler, after very early experiments¹ was introduced clinically in the early 1990s²,³ and is one of the very few recent technical innovations in echocardiography to gain quick and near-ubiquitous clinical implementation. The most widely adopted application is the registration of the mitral annular baso-apical velocity profile, at the basal septal or basal lateral left ventricular wall, which gives quantitative information on both diastolic and systolic global left ventricular function and is now part of the standard echo examination.⁴ These tissue velocities have also been shown to be useful to identify subclinical cardiomyopathies, such as hypertrophic cardiomyopathy,⁵,⁶ amyloidosis,⁷ or drug cardiotoxicity.⁸ Recent data show a strong prognostic value of solid cardiac structures by pulsed-wave Doppler, after myocardial infarction.⁹ Another established application is drug cardiotoxicity.⁸ Recent data show a strong prognostic value after myocardial infarction.⁹ Another established application is right ventricular free wall velocity as a parameter of right ventricular function.

Technically, tissue Doppler differs from standard pulsed-wave Doppler by using a low-pass frequency filter to keep low-velocity signals from tissue and to reject the high-velocity signals from blood. The detected velocities are low—even for right ventricular blood. The detected velocities are low—even for right ventricular free wall velocity as a parameter of right ventricular function.

In their study in this issue, Dhutia et al.¹⁵ analysed the accuracy of outer edge, middle line, and inner edge of tissue Doppler tracings both in vitro (comparing with an independent gold standard) and in patients (comparing with velocities calculated from M-mode and speckle tracking). They found that in vitro velocity values from the middle of the tracing corresponded best with the actual relative velocity of transducer vs. (stationary) tissue, and in patients they corresponded best with velocity calculated either by M-mode (dividing the excursion of the mitral annulus by the corresponding time interval) or by speckle tracking. Thus, measurements of the outer edge of the tracing, which are the most commonly used, are less accurate.

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(although perhaps better reproducible) than those taken from the mid-thickness of the tracing.

Some weaknesses in the methodology should be noted. The in vitro set-up of the study, where the tissue phantom is stationary, and the transducer is moved back and forth, reverses the situation in vivo and thus does not accurately replicate the ultrasound characteristics of tissue deformation. Furthermore, the true velocity in the in vitro set-up was obtained by ‘optical tracking’ (explained in their online appendix), the accuracy of which is unclear. Another important issue which this study brings up is the role of different manufacturers. Mårtensson et al. recently reported an in vitro comparison of the accuracy of six current commercially available ultrasound systems for tissue Doppler-based velocity, displacement, strain, and strain rate measurements. For velocity, they found mean differences between measured and true velocities which ranged between $0 \pm 0.4$ cm/s (corresponding to $0 \pm 5\%$ of the maximal value) in the most accurate ultrasound machine and $2.9 \pm 0.4$ cm/s ($34 \pm 3\%$ of the maximal value) in the least accurate. Such systematic errors cannot be considered trivial and are a source of concern. Differences between measurements and true values for strain and strain rate were even higher, and in addition, there were timing errors. In the present study, four different machines from four manufacturers were applied to the in vitro setting. Surprisingly, Dhutia et al. report that ‘Using the in vitro model, the M-mode, speckle tracking, and tissue Doppler (middle line) traces broadly agreed with non-ultrasound optical assessment’ (the latter being the gold standard for motion in their in vitro set-up). How to reconcile the two studies? A closer look at the data in Table 1 of shows that the agreement in the current study was actually not substantially better than in the above-cited study. Biases in the modal velocity data generated by the different machines ranged from $-1.72$ to $+0.4$ cm/s, well in line with the mean errors reported by Mårtensson et al. Thus, important lessons from the study of Dhutia et al. are that

(i) the modal velocity, which can be approximated as the mid-line of the Doppler envelope thickness, is closest to the true mean tissue velocity in the sample volume;
(ii) considerable systematic errors exist in tissue Doppler measurements.

More research is needed into the reproducibility of modal velocity measurements, and the findings from this study need to be confirmed with a more realistic tissue phantom capable of deformation. Furthermore, efforts need to be made to identify and remedy machine-related biases in such measurements. Therefore, it would be premature to call for new normal values and cut-offs for tissue Doppler based on the present study. Nevertheless, the study of Dhutia et al. commendably draws attention to a lack of consistency in current practice and should, at the very least, lead to being more clear in clinical practice as well as in research how tissue Doppler velocities actually are measured.

References