Colour tissue Doppler underestimates myocardial velocity as compared to spectral tissue Doppler: Poor reliability between both methods

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Aims Since colour tissue Doppler (CTD) has been shown to underestimate myocardial velocity, we sought to compare CTD with spectral tissue Doppler (STD) and establish agreement and corresponding thresholds for clinical applications.

Methods and results We included 52 consecutive patients with sinus rhythm referred for echocardiographic assessment. Analysis involved a commercially available echosonographer (Vivid 7, GE-Vingmed) and the Echopac system for offline assessment. Myocardial velocities were recorded by STD and CTD in a 4-chamber apical view.

CTD values were lower than those measured by STD: 6.0 ± 2.5 versus 8.2 ± 2.8 for Ea; 5.5 ± 2.3 versus 7.9 ± 2.9 for Aa, and 5.4 ± 2.0 versus 7.7 ± 2.4 for Sa (P < 0.001 for all). CTD overestimated the E/Ea: 14.7 ± 7.6 versus 10.1 ± 4.1, P < 0.001. Reliability between the two methods was low to moderate: kappa values ranged from 0.33 ± 0.10 to 0.57 ± 0.12. CTD thresholds corresponding to usual STD thresholds were calculated, but reliability was not significantly increased, except for the E/Ea ratio. By using continuous values, the ability of the Ea, Sa and E/Ea to predict the presence of heart failure in this sample was similar whatever the method.

Conclusion CTD consistently underestimates myocardial velocity values and overestimates E/Ea. A shift of thresholds between the two methods is not sufficient to obtain good agreement, except when measuring the E/Ea ratio.

KEYWORDS
Tissue Doppler; Echography; Heart failure; Diastolic function; Agreement

Introduction

Tissue Doppler is an important tool for evaluating myocardial systolic and diastolic function and for non-invasive estimation of left ventricular end-diastolic pressure.1-11 Because of the usefulness of the information gained by tissue Doppler, an increasing number of commercially available systems are proposed in this setting. At the same time and as a direct consequence, echocardiographic and Doppler assessment has become time-consuming, requiring offline analysis and use of post-processing software. Thus, 2D colour-coded tissue velocity imaging (Doppler), with its good spatial resolution and ability to measure velocity parameters simultaneously, is increasingly being used. However, small differences have been found in velocity calibration among commercially available systems,12 as well as overestimation of real velocities with use of pulsed Doppler and underestimation with colour-coded Doppler.13 Moreover, a recent study has pointed out a systematic underestimation of velocity measured by colour-coded tissue Doppler (CTD) as compared with spectral tissue Doppler (STD).14 Because different systems and various techniques of tissue velocity assessment are used, the validation of normal values5,9,11 and thresholds1-4,6-8,10,11,15 are important issues in characterizing left-ventricular function or filling pressure.

The purpose of this study was to establish whether CTD could be used instead of STD for the evaluation of myocardial systolic and diastolic function and for non-invasive estimation of left ventricular end-diastolic pressure.

Methodology

We enrolled 52 consecutive patients with sinus rhythm and stable haemodynamic conditions, who were referred for
Echocardiographic assessment in the Department of Cardiology of Beaujon Hospital (Clichy, France). The diagnosis of heart failure (23 patients) was based on the usual clinical criteria,\textsuperscript{16} Doppler echocardiographic parameters that demonstrate diastolic abnormality, with a left-ventricular ejection fraction (LVEF) $>$40\% at the time of the examination and abnormal serial brain natriuretic peptide measurements as previously described in detail\textsuperscript{17} for patients with a past history of acute heart failure. Patients with severe mitral or aortic valvular disease or abnormal lateral wall motion were excluded. All patients gave their informed consent. The study was approved by the institutional review boards of Beaujon Hospital.

Echocardiographic and Doppler measurements

Images were taken by use of a commercially available machine (Vivid 7\textsuperscript{\textregistered} GE-Vingmed, Horten, Norway) with a 3.5-MHz transducer (GE-Vingmed, Horten, Norway), and data analysis was performed offline with use of the Echopac system (GE-Vingmed, Horten, Norway). All patients were examined at rest while in the left lateral recumbent position. Left ventricular end-diastolic diameter (LVEDD) and end-diastolic interventricular wall thickness (IVW) were recorded in the parasternal long axis according to the American Society of Echocardiography guidelines.\textsuperscript{18,19} LVEF measured by Simpson or Teichholz methods, was assessed following the same guidelines.\textsuperscript{18,19}

Peak velocities of the mitral inflow were derived from pulsed Doppler recordings with the sample volume placed at the tip of the mitral leaflets. The following values were measured: peak E-wave velocity; E-wave deceleration time and peak A-wave velocity.

Velocities of myocardial parameters were recorded online (STD) and offline by use of colour-coded tissue velocity images (CTD) in a 4-chamber apical view. For both methods, velocities were measured by placing a circular sample volume of 6 mm in diameter at the lateral part of the mitral annulus. Offline CTD was decoded for quantitative analysis, and a 70-ms filter width was applied to reduce noise. The frame rate for 2D colour-coded images was always greater than 150 Hz, and special care was taken for similar placement of the sample volume, 2D image recording and maintaining Doppler angles below 30\textdegree. All recordings were performed by 2 operators (JMT, LTK). CTD and STD were performed in alternate order among patients to test the possibility of an order effect. All echocardiographic and Doppler measurements were taken at least 4 times, and results were averaged. Measurements taken with both CTD and STD were first-peak systolic velocity of the annulus (Sa), early peak diastolic velocity (Ea) and late peak diastolic velocity (Aa). Ea/Aa and E/ Ea ratios were also calculated.

Intraobserver variability for Ea, Aa and Sa assessment with CTD or STD ranged between 3% and 5%, and interobserver variability between 4% and 9%, which is in accordance with published data.\textsuperscript{4,6–8}

Statistical analysis

Statistical analysis involved use of NCSS 6.0.21 software. Continuous data were expressed as means $\pm$ standard deviation (SD) and minimum and maximum values. Values from CTD and STD assessment were compared by use of paired Student’s $t$-test. The agreement level between CTD and STD measurements was assessed by the Bland and Altman method and by plotting the difference against the mean value and its 95\% confidence interval (95\% CI).\textsuperscript{70} Spearman-rank test was used for calculating correlation. Comparison of categorical values was by kappa test. A kappa value $<$0.40 indicated low agreement, a value between 0.40 and 0.75 medium agreement, and a value $>$0.75 high agreement between the two methods. We used receiver-operating characteristic (ROC) curve analysis and maximum likelihood ratio to determine the correspondence between CTD thresholds and published STD thresholds ($Ea$ $<$8 cm/s, $Ea/Aa$ ratio $<$1, $Sa$ $<$8 cm/s, $E/Ea$ ratio $<$8 or $>$10 or $>$15).\textsuperscript{6,8–10,15} Assessing sensitivity, specificity and positive and negative predictive values of these thresholds. The CTD thresholds were chosen according to the higher ratio of maximum likelihood and used as categorical values in the analysis.

Results

Table 1 shows baseline characteristics of the studied sample. The mean age was 61 $\pm$ 13 years; 52\% were males and 44\% had chronic heart failure. No significant order effect was found between STD and CTD measurement.

Reliability of STD and CTD measurements

The mean velocity values with CTD were consistently lower than those with STD, and the $E/Ea$ ratio was consistently overestimated with CTD. The two methods did not differ in results depending on order of performance of the procedure. Bland and Altman representations of differences between methods showed an underestimation of $Ea$ (Figure 1), $Aa$ (Figure 2) and $Sa$ (Figure 3) by use of the CTD. With CTD, the $Ea/Aa$ ratio tended to be underestimated for low values and overestimated for high values with severe outliers in both cases (Figure 4). The $E/Ea$ ratio was consistently underestimated with CTD, with severe outliers for the highest values (Figure 5).

On applying the usual STD thresholds ($Ea$ $<$8 cm/s, $Ea/Aa$ $<$1, $Sa$ $<$8 cm/s, $E/Ea$ $<$8, $E/Ea$ $>$10 and $E/Ea$ $>$15) for measurement with CTD, kappa values were 0.42 $\pm$ 0.11, 0.77 $\pm$ 0.14, 0.33 $\pm$ 0.10, 0.57 $\pm$ 0.12, 0.46 $\pm$ 0.12, 0.45 $\pm$ 0.12, respectively, which shows poor agreement between the methods when used to measure categorical values.

Reliability between STD and CTD with use of specific thresholds

Table 2 shows the usual STD thresholds and the CTD thresholds predicted from STD thresholds: $Ea$ $<$3.7 cm/s, $Ea/Aa$ $<$0.9, $Sa$ $<$4.4 cm/s, $E/Ea$ $<$10.6, $E/Ea$ $>$13 and $E/Ea$ $>$22. On applying both the usual STD thresholds and specific CTD thresholds ($Ea$ $<$8 or 3.7 cm/s, $Ea/Aa$ $<$0.9, $Sa$ $<$8 or 4.4 cm/s, $E/Ea$ $<$8 or 10.6, $E/Ea$ $>$10 or 13 and $E/Ea$ $>$15 or 22) for measurement in the same patient, kappa values were 0.23 $\pm$ 0.11, 0.59 $\pm$ 0.13, 0.54 $\pm$ 0.13, 0.92 $\pm$ 0.14, 0.76 $\pm$ 0.14, and 0.56 $\pm$ 0.14, respectively, which shows poor agreement between methods in assessing $Ea$, moderate agreement in assessing $Ea/Aa$ ratio and $Sa$ and moderate or good agreement in calculating $E/Ea$ ratio, especially for low values of the ratio ($E/E_{STD-a}$ $<$8 or $E/E_{CTD-a}$ $<$10.6).

Relation between STD or CTD and the presence of chronic heart failure

When STD or CTD values were used as continuous variables, the relation between $Ea$, $Sa$ or $E/Ea$ ratio with the presence of chronic heart failure was similar between the 2 methods. Areas under the ROC curves for STD versus CTD were 0.86 $\pm$ 0.20 versus 0.88 $\pm$ 0.20 for $Ea$; 0.77 $\pm$ 0.19 versus 0.80 $\pm$ 0.19 for $Sa$; and 0.82 $\pm$ 0.19 versus 0.85 $\pm$ 0.19 for $E/Ea$ ratio.
Discussion

This study confirms the consistent underestimation of velocity values of myocardial parameters with CTD as compared with STD. Moreover, it points out the poor agreement between the two methods when used to assess categorical values. However, estimating CTD thresholds on the basis of STD thresholds gave moderate agreement in assessing Ea/Aa ratio and Sa and moderate or good agreement in calculating Ea/Ea ratio, especially for low values of the ratio (E/Ea_CTDA < 8 or E/Ea_STD < 10.6).

Several explanations are possible for the consistent underestimation of myocardial velocities by use of CTD. First, pulsed Doppler records peak velocity, whereas offline colour-decoded Doppler measures mean velocity. Second, the use of filter width to reduce noise is associated with a systematic underestimation of velocity, especially for rapid myocardial movements such as the isovolumic periods. Therefore, the larger the filter width, the greater the underestimation, varying according to the duration of the left ventricular diastolic or systolic period. In our study, we chose a filter width of 70 ms, which is important in terms of reducing noise in critically ill patients with very low myocardial velocity values as compared to normal subjects. Moreover, this filter width was predefined by the manufacturer of our echosonographer. A shorter filter width would have led to a smaller underestimation and different thresholds under other conditions. As well, this filter width

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Baseline characteristics of the study sample</th>
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<tbody>
<tr>
<td>Age, years</td>
<td>61 ± 13</td>
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<tr>
<td>Sex (male), %</td>
<td>52</td>
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<tr>
<td>BSA, m²</td>
<td>1.8 ± 0.5</td>
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<td>Coronary disease, %</td>
<td>27</td>
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<td>CHF, %</td>
<td>44</td>
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<td>NYHA (I/II/III/IV), N*</td>
<td>7/15/1/0</td>
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<tr>
<td>Heart rate, bpm</td>
<td>70 ± 14</td>
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<tr>
<td>LVEF, %</td>
<td>53 ± 20</td>
</tr>
<tr>
<td>LVEDD, mm</td>
<td>54 ± 11</td>
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<tr>
<td>IVW, mm</td>
<td>10 ± 2</td>
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<tr>
<td>E, cm/s</td>
<td>74.6 ± 19.4</td>
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<tr>
<td>A, cm/s</td>
<td>69.2 ± 23.0</td>
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<tr>
<td>DT, ms</td>
<td>205 ± 51</td>
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<tr>
<td>Ea, cm/s</td>
<td>8.2 ± 2.8 (3.8–16.0)</td>
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<tr>
<td>Aa, cm/s</td>
<td>7.9 ± 2.9 (3.0–15.0)</td>
</tr>
<tr>
<td>Ea/Aa ratio</td>
<td>1.1 ± 0.5 (0.3–2.6)</td>
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<tr>
<td>Sa, cm/s</td>
<td>7.7 ± 2.4 (3.3–13.1)</td>
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<tr>
<td>E/Ea ratio</td>
<td>10.1 ± 4.1 (4.5–20.1)</td>
</tr>
</tbody>
</table>

* Number of patients with chronic heart failure and New York Heart Association classification I or II or III or IV. BSA, body surface area; CHF, chronic heart failure; LVEF, left ventricular ejection fraction; LVDD, left ventricular end-diastolic diameter; IVW, interventricular wall thickness; DT, E wave deceleration time; STD, online spectral tissue Doppler assessment of tissue velocities; STD, offline colour tissue Doppler assessment of tissue velocities; Sa, first-peak systolic velocity of the mitral annulus; Ea, early peak diastolic velocity of the mitral annulus; Aa, late peak diastolic velocity of the mitral annulus.

Figure 1 Bland and Altman representation of the reproducibility of the early diastolic peak velocity of the lateral part of the mitral annulus (Ea) assessed by spectral tissue Doppler (STD) or colour tissue Doppler (CTD).

Figure 2 Bland and Altman representation of the reproducibility of the late diastolic peak velocity of the lateral part of the mitral annulus (Aa) assessed by STD or CTD.
is not recommended for rapid myocardial movements such as during isovolumic periods.\textsuperscript{21}

Our findings highlight those of several recent published studies showing that CTD with comparable systems (GE-VingMed: System Five,\textsuperscript{1,2,11} VIVID 7,\textsuperscript{9,11} Echopac\textsuperscript{1,2}) revealed a high proportion of left ventricular diastolic or systolic dysfunction and elevated left ventricular filling pressure in hypertensive subjects or the general population.\textsuperscript{1,2,9} Wang \textit{et al.}\textsuperscript{2} demonstrated the prognostic value of CTD-determined $E_a$ in a hypertensive population with left-ventricular hypertrophy. As expected, the threshold for $E_a$ ($<3.5$ cm/s) was well below the usual $8$ cm/s\textsuperscript{15} for STD, which confirms the underestimation of myocardial velocities with CTD, but is very close to the value we suggest ($<3.7$ cm/s). Moreover, this study showed that 41.1\% of patients with an abnormal relaxation pattern had an $E/E_a > 15$. The use of a threshold of 22 instead of 15 (with a similar filter width and frame rate), according to the underestimation of the method, should have decreased the prevalence of high left-ventricular filling pressure. However, this explanation is probably not sufficient, because De Sutter \textit{et al.}\textsuperscript{9} found a high prevalence of hypertension with a high $E/E_a$ ratio on measurement with STD. The authors suggested that a high threshold should be adapted for the same method (STD) for a population not studied in the original articles by Nagueh \textit{et al.}\textsuperscript{6–8,10} and Ommen \textit{et al.}\textsuperscript{4} These authors showed that the higher the $E/E_a$ threshold, the lower the sensitivity (increasing the number of false-negative results) and the higher the specificity (decreasing the number of false-positive results) of the method used to detect disease. Because sensitivity and specificity depend on the population studied, these are presently unknown with CTD and STD in populations with very low prevalence of or lacking symptomatic heart failure, and the ability of an $E/E_a$ ratio $> 10$ or 15 to predict high left-ventricular filling pressure remains to be demonstrated in these conditions. Moreover, if the $E/E_a$ ratio remains the best predictor of elevated left-ventricular filling pressure for patients without cardiac disease, the accuracy of an $E/E_a$ ratio $> 15$ is especially low (sensitivity 74\%, specificity 72\%) as a predictor of end-diastolic pressure over 15 mmHg.\textsuperscript{10} Finally, Yu \textit{et al.}\textsuperscript{11} showed that a value of $S_a < 4.4$ cm/s (mean of 6 basal segments), determined by use of CTD (Vivid 7 and Echopac system as in the present study) was predictive of systolic dysfunction regardless of ejection fraction, this threshold being identical to our suggested threshold on the basal lateral segment.

Our study contains several limitations. First, the differences between methods could be explained in part by the percentage of error due to reproducibility. Second, the differences between methods in terms of angle modification are unlikely because differences should have been of a similar range compared with reproducibility and not multiplied by a factor of 5–10. Moreover, because lateral velocities are higher than medial velocities,\textsuperscript{7} our results can probably not be applied to medial assessment of tissue velocities. Finally, the characteristics of the sample could have influenced the results.

Regarding the assessment of left ventricular end-diastolic pressure, the lack of an invasive validation is clearly a limitation to determine which method is the most accurate. However, because in this study each patient was his own control, and the lack of invasive validation cannot be an explanation for these discrepancies.

In summary, because of the usefulness of the assessment of myocardial velocity parameters in a wide range of cardiovascular disease and because this assessment is time-consuming for patients and staff, offline analysis and the use of post-processing software such as that with CTD are required. We have demonstrated in well-defined technical...
conditions that CTD was associated with an important underestimation of velocity values as compared with STD, which led to misclassification of disease. Moreover, our study points out the poor agreement between the two methods when assessing categorical values and the inability to increase this agreement by using higher thresholds with CTD in assessing Ea/Aa ratio and Sa and Ea. However, despite the magnitude of differences between CTD and STD in measuring myocardial velocities and the technical limitations of each method, our specific thresholds for estimation of left ventricular end-diastolic pressure by use of CTD could be useful. However, further studies are needed to establish the relation between CTD parameters and invasive parameters of myocardial systolic and diastolic functions and left ventricular end-diastolic pressure.

References


