Doppler Estimation of Left Ventricular Filling Pressures in Patients With Hypertrophic Cardiomyopathy

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Background—Conventional Doppler parameters are unreliable for estimating left ventricular (LV) filling pressures in hypertrophic cardiomyopathy (HCM). This study was undertaken to evaluate flow propagation velocity by color M-mode and early diastolic annular velocity (Ea) by tissue Doppler 2 new indices of LV relaxation, combined with mitral E velocity for estimation of filling pressures in HCM.

Methods and Results—Thirty-five HCM patients (52±15 years) underwent LV catheterization simultaneously with 2-dimensional and Doppler echocardiography. Pulsed Doppler echocardiography of mitral and pulmonary venous flows was obtained along with flow propagation velocity and Ea. LV preA pressure had weak or no relations with mitral, pulmonary venous velocities and atrial volumes. In contrast, preA pressure related strongly to E velocity/flow propagation velocity (r=0.67; SEE=4) and E/Ea (r=0.76; SEE=3.4). In 17 patients with repeat measurements, preA pressure changes were well detected by measuring E velocity/flow propagation velocity (r=0.68; P=0.01) or E/Ea (r=0.8; P<0.001). PreA pressure estimation with these 2 methods was tested prospectively in 17 additional HCM patients with good results (E velocity/flow propagation velocity, r=0.76; E/Ea, r=0.82).

Conclusions—LV filling pressures can be estimated with reasonable accuracy in HCM patients by measuring E velocity/flow propagation velocity or E/Ea. These ratios also track changes in filling pressures. (Circulation. 1999;99:254-261.)

Key Words: echocardiography ■ pressure ■ ultrasonics, Doppler ■ hypertrophy ■ cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is a genetic disease that frequently presents with dyspnea. Although left ventricular outflow tract (LVOT) obstruction is not always there, diastolic dysfunction is almost universally present. Thus, a noninvasive method for determination of LV filling pressures in HCM is highly desirable. Although echocardiography has been successfully used to estimate filling pressures in a variety of cardiac disorders, current methods are unreliable with normal systolic function, particularly in HCM. These results are related in part to the impaired relaxation-dominant influence on mitral inflow that overshadows the effects of increased filling pressures. Thus, mitral peak velocity of early filling (E velocity) combined with a load-independent index of relaxation may improve Doppler estimation of filling pressures. There are currently 2 relaxation indices that appear less preload-dependent: early flow propagation velocity by color M-mode and early diastolic annular velocity (Ea) by tissue Doppler (TD). These indices may correct for the influence of relaxation on mitral E velocity. We and others have noted that mitral E velocity/flow propagation velocity relates well to filling pressures. Similarly, we showed recently that E/Ea predicts filling pressures well throughout a wide range of systolic performance. It is currently unknown whether E velocity/flow propagation velocity or E/Ea can be applied in HCM. Our purpose was, therefore, to determine the role of these new noninvasive relaxation parameters in estimating filling pressures in HCM patients.

Methods

The protocol was approved by the institutional review boards of Methodist Hospital and Baylor College of Medicine, and all patients gave written informed consent before participation. The group was composed of 35 HCM patients enrolled for ethanol septal reduction therapy. Patients had asymmetrical septal hypertrophy with septal thickness ≥1.5 cm, and septum/posterior wall thickness ≥1.3. They were all dyspneic, and most were in NYHA functional class III or IV. Inclusion criteria were normal sinus rhythm, absence of mitral stenosis or prosthetic mitral valve, and adequate Doppler measurements. All had simultaneous Doppler (including color M-mode and TD) and LV pressure measurements.

Echocardiographic Studies

Patients were imaged in supine position with the Acuson XP-128 ultrasound system equipped with a multifrequency transducer and TD. From the apical view, the pulsed Doppler sample volume was placed at mitral valve annulus then at tips, and 5 to 10 cardiac cycles...
were recorded at each site during normal respiration. The cursor was placed between LV outflow and mitral inflow to record isovolumic relaxation time (IVRT). Pulmonary venous (PV) flow was recorded from the right vein with color Doppler guidance. With the use of color Doppler echocardiography, the M-mode cursor was positioned within the center of the mitral inflow stream, and M-mode recording of the early flow propagation velocity into the LV was obtained. Baseline shift was performed as needed to obtain a distinct color border of the propagation velocity that extended into the distal portion of the LV cavity. The TD program was set to pulsed-wave mode (30 cm/s), with gain adjusted to minimize background noise. From the 4-chamber view, a 5-mm sample volume was placed at the lateral border of the mitral annulus and 5 to 10 cycles were recorded. Studies were stored on 1/2-in VHS videotape for later playback and analysis.

Echocardiographic Analysis

All measurements were made by an observer blinded to hemodynamic data on an off-line analysis station (Digisonics EC500) with 2-dimensional and Doppler measurement software. Left atrial23 and LV volumes were derived with the discs method. Mitral inflow was measured and traces were obtained for the following: peak velocity of early (E) and late (A) filling, acceleration time of E (AT-E), deceleration times of E (DT-E) and A (DT-A), and atrial filling fraction (AFF). IVRT was measured as previously described. Pulmonary venous (PV) inflow was analyzed for peak velocity and velocity time integral (VTI) of systolic (S), diastolic (D), and atrial reversal waves (Ar; Figure 1). Systolic filling fraction (SFF) was calculated as follows: S/VTI/total forward flow VTI. Mitral A velocity duration at the annulus and Ar duration were measured. Subsequently, Ar-A duration was derived.6 The flow propagation velocity (cm/s) was measured as the slope of the linear component of the color border produced by propagation of E velocity into the LV, past the mitral valve tips14,20 (Figure 1). Mitral E velocity/flow propagation velocity was calculated.20,21 The following measurements were made from TD tracings (Figure 1): early (Ea) and late (Aa) diastolic annular velocities, Ea/Aa, and mitral E/Ea ratios.5

Pressure Measurements

A 7F pigtail catheter was used for LV pressure measurements. Medex transducers (fluid column length, 1.88 in; observed natural frequency, 105 Hz) were balanced before acquisition of hemodynamic data with zero level at mid axillary line. Baseline pressure measurements were acquired before coronary angiography, and none of the patients had ventriculography. LV diastolic pressures measured were as follows: minimal pressure, preA, and end diastolic pressure (LVEDP; Figure 1B). PreA pressure was taken before the pressure increase due to atrial contraction, and LVEDP was determined before the rise in systolic pressure (at end-expiration with the average of 3 cycles). After septal infarction, patients received meperidine hydrochloride (Demerol) or morphine. Seventeen patients in normal sinus rhythm had repeat measurements of filling pressures simultaneous with Doppler echocardiography.

Test Population

The equations derived to estimate filling pressures were tested in 17 consecutive patients (9 of them women, 47±18 years; age range, 19 to 74 years). Doppler and pressure data were obtained simultaneously. Patients studied had the same inclusion and exclusion criteria as the initial group. Doppler measurements and calculations were made without knowledge of hemodynamics.
TABLE 1. LV Filling Pressures and Echocardiography-Doppler Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± SD (Range)</th>
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<tbody>
<tr>
<td>PreA pressure, mm Hg</td>
<td>14 ± 5 (4–30)</td>
</tr>
<tr>
<td>LVEDP, mm Hg</td>
<td>23 ± 6 (10–38)</td>
</tr>
<tr>
<td>LV minimal pressure, mm Hg</td>
<td>9 ± 5 (0–21)</td>
</tr>
<tr>
<td>LA maximal volume, mL</td>
<td>62 ± 28 (24–160)</td>
</tr>
<tr>
<td>LA minimal volume, mL</td>
<td>40 ± 21 (12–100)</td>
</tr>
<tr>
<td>LA-EF, %</td>
<td>40 ± 17 (20–84)</td>
</tr>
<tr>
<td>E, cm/s</td>
<td>64 ± 16 (30–97)</td>
</tr>
<tr>
<td>A, cm/s</td>
<td>72 ± 25 (23–132)</td>
</tr>
<tr>
<td>E/A</td>
<td>1 ± 0.6 (0.4–3.5)</td>
</tr>
<tr>
<td>DT, ms</td>
<td>238 ± 103 (82–458)</td>
</tr>
<tr>
<td>Flow propagation velocity, cm/s</td>
<td>35 ± 12 (17–67)</td>
</tr>
<tr>
<td>Ea, cm/s</td>
<td>6.7 ± 1.7 (4–11)</td>
</tr>
<tr>
<td>PV SFF, %</td>
<td>50 ± 13 (25–71)</td>
</tr>
<tr>
<td>PV Ar-A, ms</td>
<td>25 ± 30 (19–98)</td>
</tr>
</tbody>
</table>

LA indicates left atrium; EF, emptying fraction; Ar-A, PV atrial reversal velocity duration—mitral A duration. Values are mean±SD (range).

Statistical Analysis
Linear regression analysis was used to correlate continuous variables with each other. Agreement between Doppler-estimated and catheter-measured pressures was evaluated by plotting the difference against the mean value of the 2 measurements (Bland-Altman plots). We estimated a correlation coefficient between Doppler parameters and LV diastolic pressures of ~0.6 to 0.7, with a power of 0.9 at α=0.05. Accordingly, our sample size of 35 was adequate to detect such a relation (calculated sample size of 25 at r=0.6). Significance was set at P<0.05.

Results
Mean age was 52±15 years (range, 24 to 83 years; 19 men, heart rate 71±9 bpm; range, 52 to 86 bpm). All but 4 had a LVOT gradient due to systolic anterior motion of the mitral valve at rest (56±29 mm Hg; range, 15 to 120 mm Hg). They all had hyperdynamic ventricles, but none had severe mitral regurgitation. Satisfactory mitral inflow signals and mitral annulus TD recordings were obtained in 35 patients, flow propagation velocity in 31, and PV flow in 23.

Correlation of Filling Pressures with Doppler Parameters
Table 1 summarizes the LV pressures and Doppler parameters for the study group. A preA pressure of 14±5 mm Hg (range, 4 to 30 mm Hg) did not correlate with any of the left atrial volumes. Significant weak correlations were present with mitral E velocity (r=0.4; P=0.02) and E/A ratio (r=0.33; P=0.04; Table 2) with no relations to other trans-mitral flow parameters (Figure 2). None of the PV velocities or VTIs including SFF related significantly with preA pressure (Figure 2). Despite the poor relations of conventional Doppler indices with preA pressure, all had cutoff thresholds beyond which they were specific for pressure >15 mm Hg (specificity of E/A ≥1.1 and SFF ≥40% of 83% to a specificity of 92% for IVRT ≤70 ms). Ar-A duration (PV atrial reversal velocity duration minus mitral A duration) exhibited a significant relation with LVEDP (r=0.53, P<0.01; Figure 3). Applying a previous equation9 from our laboratory to predict preA pressure in non-HCM patients resulted in a weak relation (r=0.35, P=0.03). Likewise, a previous equation9 derived in our laboratory to estimate LVEDP in non-HCM patients demonstrated no relation with the measured LVEDP (r=0.14).

Flow propagation velocity had no significant relation to preA pressure but related significantly to LV minimal pressure (r=−0.5, P<0.01, Figure 4). E velocity/flow propagation velocity demonstrated a strong relation with preA pressure (r=0.67; SEE=4 mm Hg; P=0.02; Figure 5). Ea demonstrated a weak relation with preA pressure (r=−0.35, P=0.01) and a strong relation with LV minimal pressure (r=−0.73, P<0.001, Figure 4). Ea also had a significant inverse correlation with the LV end systolic volume (r=−0.55, P=0.02). E/Ea related well to preA pressure (r=0.76; SEE=3.4 mm Hg; P<0.001; Figure 6). PreA pressure could be estimated with E velocity/flow propagation velocity as follows: 4.5+[5.28×(E velocity/propagation velocity)], and E/Ea could be estimated as 3.2+[1.1×(E/Ea)]. The mean difference between the Doppler estimate and catheter measurement was 0±3.9 mm Hg (range, −9 to 8 mm Hg) when E velocity/flow propagation velocity was used, and 0±3.3 mm Hg (range, −8 to 6.8 mm Hg) with E/Ea (Bland-Altman plots in Figures 5 and 6). The correlation between the new derived indices and preA pressure was further examined in 11 patients with E/A <1 and DT >250 ms (severely impaired relaxation); E velocity/flow propagation velocity and E/Ea still related well to preA pressure (r=0.62 and 0.72, respectively).

Detection of Filling Pressures Changes
Ten patients (10 of 35) developed complete heart block and were pacemaker-dependent immediately after ethanol-

TABLE 2. Correlation Between Echocardiographic Parameters and PreA Pressure

<table>
<thead>
<tr>
<th>Parameter</th>
<th>r</th>
<th>PreA Parameters</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>LA maximal volume, mL</td>
<td>0.3</td>
<td>PV systolic velocity, cm/s</td>
<td>−0.12</td>
</tr>
<tr>
<td>LA minimal volume, mL</td>
<td>0.3</td>
<td>PV diastolic velocity, cm/s</td>
<td>0.26</td>
</tr>
<tr>
<td>LA-EF, %</td>
<td>−0.12</td>
<td>PV systolic VTI, cm</td>
<td>−0.17</td>
</tr>
<tr>
<td>E, cm/s</td>
<td>0.4*</td>
<td>PV diastolic VTI, cm</td>
<td>0.4</td>
</tr>
<tr>
<td>A, cm/s</td>
<td>0.05</td>
<td>SFF</td>
<td>−0.19</td>
</tr>
<tr>
<td>E/A</td>
<td>0.33*</td>
<td>Ar-A, ms</td>
<td>0.46*</td>
</tr>
<tr>
<td>AT, ms</td>
<td>−0.26</td>
<td>DT, ms</td>
<td>−0.17</td>
</tr>
<tr>
<td>AFF</td>
<td>0.04</td>
<td>DT-A, ms</td>
<td>0.03</td>
</tr>
<tr>
<td>VRT, ms</td>
<td>−0.2</td>
<td>Early propagation velocity, cm/s</td>
<td>0.26</td>
</tr>
<tr>
<td>E/early propagation velocity</td>
<td>0.67*</td>
<td>Ea, cm/s</td>
<td>−0.35*</td>
</tr>
<tr>
<td>Aa, cm/s</td>
<td>−0.26</td>
<td>Ea/Aa</td>
<td>0.09</td>
</tr>
</tbody>
</table>
| E/Ea | 0.76* | AT indicates acceleration time-E; DT-A, deceleration time-A; other abbreviations as Table 1. *P<0.05.

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induced septal infarction; 8 of 35 did not undergo repeated measurements of pressures. Accordingly, only 17 patients in normal sinus rhythm underwent repeated simultaneous measurements of LV pressures with Doppler echocardiography. No significant relations were present between preA pressure changes and changes in AT-E, DT-E, DT-A, AFF, and IVRT. Weak but insignificant relations were noted with changes in A velocity ($r = -0.43; P = 0.1$), E velocity/A velocity ($r = 0.44; P = 0.09$), and Ea ($r = -0.4; P = 0.11$). Significant relations were present, however, between preA pressure changes and E velocity changes ($r = 0.56; P = 0.01$), E velocity/flow propagation velocity ($r = 0.68; P = 0.01$), and E/Ea ($r = 0.8; \text{SEE} = 3.6 \text{ mm Hg}; P < 0.001$; Figure 7). Five of the 6 patients with increases in preA pressure had increments in E/Ea $\geq 2$. In 1 patient, preA pressure was reduced by 5 mm Hg with diuretics; E/Ea decreased by 6.1, predicting a pressure reduction of 6 to 7 mm Hg (Bland-Altman plot in Figure 7).

Test Population

The equations derived with the use of E velocity/flow propagation velocity and E/Ea in the initial group were tested prospectively in the test population. Flow propagation velocity was satisfactorily measured in 11 patients (in 6, the quality of the 2-dimensional traces was not adequate because of patients’ supine position on the catheterization table), whereas Ea was ascertained in all 17. Estimation of preA pressure was possible with either method (Figure 8). With the E velocity/flow propagation velocity, the correlation coefficient was 0.76, and the mean difference between Doppler-estimated and cather-measured pressures was $-0.5 \pm 5$ mm Hg. Applying E/Ea, the correlation coefficient was 0.82 and the mean difference was $-0.7 \pm 3$ mm Hg between Doppler-estimated and catheter-measured pressures. When the 52 patients were combined, E/Ea $\geq 10$ had the best combination of sensitivity (92%) and specificity (85%) for preA pressure $> 15$ mm Hg (area under receiver operating characteristic curve = 0.91; Figure 9). For E velocity/flow propagation velocity, a ratio $\geq 1.8$ had a sensitivity of 79% with a specificity of 77%. A lower ratio ($\geq 1.6$) had a higher sensitivity (95%) with a lower specificity (54%), whereas a ratio $\geq 2$ was more specific (88%) but less sensitive (68%).

Discussion

This study demonstrates for the first time the role of 2 new LV relaxation indices, the flow propagation velocity and Ea, in estimation of LV filling pressures in patients with HCM. Conventional mitral inflow and PV velocities had weak or no relation to filling pressures. However, mitral E velocity, corrected for the influence of LV relaxation with the use of either of the 2 new indices, related well to preA pressure. Figure 1 is from a patient with a preA pressure of 24 mm Hg. E velocity/flow propagation velocity (2.7) predicted a 19 mm Hg pressure. Likewise, E/Ea (16.7) predicted a 22 mm Hg pressure. Notice that the E/A ratio is $< 1$ and that
the DT is prolonged, findings usually associated with normal filling pressures in non-HCM patients.

Use of Conventional Doppler in the Estimation of Filling Pressures in HCM Patients

Mitral and PV velocities are currently used in patients with systolic dysfunction to estimate LV filling pressures with reasonable accuracy.\(^2\)\(^-\)\(^11\) Recently, Nishimura et al\(^1\)\(^3\) reported that HCM patients present a particular challenge whereby conventional Doppler parameters (DT-E and E/A) are poorly predictive of filling pressures. Our findings further corroborate these observations. In the HCM patients in our study, among the mitral inflow parameters, only the E velocity and E/A ratio had significant but weak correlations with filling pressures. DT-E, DT-A, IVRT, and AFF had no correlation with LV diastolic pressures. Despite high filling pressures, many patients had prolonged DT-E, E/A, and a high AFF. Because preload and relaxation both influence mitral E velocity,\(^2\)\(^4\) this poor correlation was present in HCM, in which relaxation is severely impaired. Atrial volumes have been previously found to relate to filling pressures.\(^6\) Accordingly, they were evaluated; however, the results were still poor. Because many HCM patients initially have left atrial hypertension in response to severely impaired relaxation, atrial dilatation is frequently present regardless of atrial pressures.

The PV systolic and diastolic velocities also related poorly to filling pressures, with SFF having no relation to the LV diastolic pressures measured in the present study. A similar observation was recently made by Yamamoto et al\(^1\)\(^2\) in non-HCM patients with normal ejection fractions (\(r=-0.29\)). In our series, a number of the patients with high filling pressures had a SFF >50%. The following may explain why the SFF remains normal in the presence of elevated filling pressures in patients with HCM. The PV systolic and diastolic antegrade flows occur in response to pressure gradients between the PV and the left atrium and are modulated by left atrial compliance and relaxation, in addition to LV relaxation and systolic function.\(^3\) In HCM patients during systole, a relatively higher pressure gradient (compared with diastole) is established between the PV and the left atrium, in part because of a normal (or increased) mitral annular descent that occurs with the hyperdynamic LV function and possibly also a hyperdynamic function and relaxation of the atrium, thus preserving systolic (S1 and S2) antegrade flow. In contrast, during diastole a relatively lower pressure gradient between the veins and the left atrium occurs because of the markedly prolonged LV relaxation, which leads to reduced atrial emptying in early diastole and, consequently, reduced PV diastolic antegrade flow.
The Ar-A duration was the only variable that had a relation to LVEDP. However, the correlation was still weak and with large spread (SEE=6 to 7 mm Hg). This may be related in part to the presence of only a few patients with LVEDP <15 mm Hg and Ar-A duration ≤0. Accordingly, it is possible that if we had had a larger number of HCM patients, the relation between Ar-A and LVEDP could have been improved. In summary, the mitral and PV flow variables are helpful only if they show patterns consistent with high filling pressures. However, these conventional parameters are not sensitive in patients with HCM.

Application of the New LV Relaxation Indices in HCM Patients

Transmitral flow follows the development of an atrioventricular pressure gradient. This gradient is determined by the left atrial V wave and the LV minimal pressure. In early diastole, the ventricular pressure normally decreases rapidly before the mitral valve opens, creating a positive pressure gradient, whereas in HCM it does so at a slower rate. This is related to contraction load (due to LVOT obstruction) delaying LV relaxation.25 Also, the disparity between the myocardial mass and capillary density and the inadequate vasodilator reserve are contributing factors.25 However, the energy stored during systole provides restoring forces that allow HCM patients to develop low diastolic pressures rapidly and to “suck” blood from the atrium. In HCM, these forces can be greater than those in healthy subjects given the small end-systolic volumes that are frequently present in HCM patients with small and hyperdynamic hearts. Thus, some HCM patients can have normal filling pressures.13 Given the complex interactions between these variables in HCM, we explored 2 relatively load-independent LV relaxation indices, flow propagation velocity and Ea. The propagation velocity relates inversely with \( t^{14-16} \) and LV minimal pressure.25,26 We demonstrated its relation to LV minimal pressure in HCM patients. E velocity/flow propagation velocity has related well to LV filling pressures in previous investigations, in sinus rhythm21 and in atrial fibrillation.20 The correlations in both studies were similar to the one in the present investigation (\( r=0.8^{21} \) and \( r=0.65^{20} \)). The higher correlation observed in a previous study21 may be related to the inclusion of patients with systolic dysfunction, in which the mitral E velocity alone relates better to filling pressures. In the present study, acute changes in preA pressure did not alter the propagation velocity, further demonstrating the insensitivity of this index to preload. In contrast, these preload changes produced similar directional changes in mitral E velocity. The propagation velocity corrected for the influence of LV relaxation on the E velocity and, thus, unmasked the pressure changes.

Ea relates inversely to \( \tau^{26,27} \) has no relation to LVEF,18 and identifies patients with a pseudonormal mitral inflow pattern.18 We extend the aforementioned observations and show the strong inverse relation between LV minimal pressure and Ea.
general, the smaller the end systolic volume, the greater the LV elastic recoil and the lower the LV minimal pressure and the faster the mitral annuluar basal displacement in early diastole. Sohn et al.27 have shown no Ea changes with saline infusion or nitroglycerin, although these interventions induced changes in E velocity and DT-E. We previously evaluated this index in conjunction with E velocity in non-HCM patients and found that E/Ea relates well to filling pressures across a wide range of pressures.18 This investigation demonstrates that E/Ea can be applied to estimate LV filling pressures in HCM patients in a similar manner. In general, preA pressure and pressure changes had higher correlations with E/Ea than with E velocity/propagation velocity.

**Study Limitations**

Satisfactory PV velocity recordings were obtained in 23 patients (66%). This success rate is lower than what we experience routinely in our laboratory and was probably the result of examining patients in the supine position in the catheterization laboratory. Although a lateral decubitus position could have provided a higher yield, imaging in this position is mostly feasible outside the catheterization laboratory and would have precluded the simultaneous acquisition of pressure and Doppler data. Except for Ar-A, it is unlikely that a higher success rate of PV velocity recordings would have altered the results, given the poor relation between these velocities and filling pressures. In contrast, flow propagation velocity and Ea were recorded in the majority of patients. Annular velocities can be recorded at multiple corners of the annulus. We measured Ea at the lateral corner because we previously noted that the lateral annulus Ea is higher and more reproducible than that of the septal Ea,18 particularly in HCM patients who often have low septal Eas. Furthermore, our study design included repeat measurements of Ea after septal infarction, and, therefore, septal velocities then will reflect more regional than global diastolic function in comparison to lateral annulus Ea.

There are currently many methods that can be used to measure the early flow propagation velocity. One involves the use of computer analysis to determine either time intervals or slope along the color flow distribution.14,16 Another approach determines the slope of a line connecting 2 points, the point of maximal velocity around the mitral orifice and the point at which this velocity decreases to 70% of its initial value.15 A modified approach used by our group and others involves baseline shift until a distinct color border is obtained and then measurement of the slope of its most linear component past the valve leaflets. At times, blurred wave front signals make it difficult to determine the margin and is a limitation for this methodology. This may explain the better results with E/Ea. Temporal and regional relaxation differences are common in patients with HCM25 and contribute to the diastolic dysfunction, limiting to some degree the application of a single measurement of flow propagation and Ea as parameters of LV relaxation. The 95% confidence limits of the Doppler estimates of filling pressures with either of the 2 new methods were wide and limited the accuracy of an individual estimate (particularly using the flow propagation velocity). Nevertheless, the new methods described in this investigation, which use cutoff values with a high specificity, can provide a good separation between HCM patients with high filling pressures and those with low filling pressures and can detect changes in filling pressures after interventions. Therefore, this new methodology represents a measurable advance in the noninvasive estimation of LV filling pressures in HCM patients.
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References
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