Hypertrophic Cardiomyopathy

Myocardial Velocity Gradient as a Noninvasively Determined Index of Left Ventricular Diastolic Dysfunction in Patients With Hypertrophic Cardiomyopathy

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OBJECTIVES We investigated the utility of the peak negative myocardial velocity gradient (MVG) derived from tissue Doppler imaging (TDI) for evaluation of diastolic dysfunction in patients with hypertrophic cardiomyopathy (HCM).

BACKGROUND Hypertrophic cardiomyopathy is characterized by impaired diastolic function with abnormal stiffness and prolonged relaxation. However, it remains difficult to evaluate these defects noninvasively.

METHODS Both TDI and conventional echocardiography were performed in 36 patients with HCM and in 47 control subjects. Left ventricular (LV) pressure was measured simultaneously in all HCM patients and in 26 controls.

RESULTS The peak negative MVG occurred soon after the isovolumic relaxation period during the initial phase of rapid filling (auxotonic relaxation). It was significantly smaller in HCM patients than in control subjects (2.32 ± 0.52/s vs. 4.82 ± 1.15/s, p < 0.0001); the cutoff value for differentiation between all HCM patients and 47 normal individuals was determined as 3.2/s. Both the left ventricular end-diastolic pressure (LVEDP) (19.6 ± 6.1 mm Hg vs. 6.5 ± 1.7 mm Hg, p < 0.0001) and the time constant of LV pressure decay during isovolumic diastole (tau) (44.0 ± 6.7 ms vs. 32.1 ± 5.5 ms, p < 0.0001) were increased in HCM patients compared with controls. The peak negative MVG was negatively correlated with both LVEDP (r = −0.75, p < 0.0001) and tau (r = −0.58, p < 0.0001).

CONCLUSIONS A reduced peak negative MVG reflects both prolonged relaxation and elevated LVEDP. The peak negative MVG might thus provide a noninvasive index of diastolic function, yielding unique information about auxotonic relaxation in patients with HCM. (J Am Coll Cardiol 2003;42:278–85) © 2003 by the American College of Cardiology Foundation

Diastolic dysfunction is an important feature of the pathophysiology of hypertrophic cardiomyopathy (HCM), which is characterized by abnormal stiffness of the left ventricle and consequent impaired ventricular filling and prolonged relaxation (1). These abnormalities result in an increased left ventricular end-diastolic pressure (LVEDP), pulmonary congestion, and dyspnea, which are the most common symptoms of HCM. However, precise indexes for assessment of diastolic function in individuals with HCM have not been established. A diagnosis of diastolic dysfunction requires evidence of abnormal left ventricular (LV) relaxation and filling, diastolic distensibility, and diastolic stiffness (2). Conventional clinical evaluation of LV relaxation involves determination of the peak negative dP/dt (first derivative of left ventricular pressure) or the time constant of left ventricular pressure decay (tau). The LV diastolic distensibility and stiffness, which lead to an increased LVEDP or pulmonary venous pressure, are evaluated by assessment of the pressure-volume curve through an invasive procedure. Although Doppler echocardiography has been widely used for noninvasive assessment of diastolic function, both the reliability and the reproducibility of this approach remain controversial given that LV filling patterns are influenced by preload (3,4).

Tissue Doppler imaging (TDI) allows quantitative assessment of regional wall motion and intramural myocardial velocity by detecting successive phase shifts of the ultrasound signal reflected from the myocardium throughout the cardiac cycle (5–7). The myocardial velocity gradient (MVG) derived from TDI was introduced as an ultrasonic index of myocardial function that is independent of translational motion (8,9). The MVG is defined as the difference in myocardial velocity between the endocardium and the...
epicardium divided by myocardial wall thickness, and therefore it reflects the dynamics of myocardial thickening and thinning (8–10).

The peak negative MVG provides an accurate estimate of LV relaxation (11–13) and, unlike conventional Doppler echocardiography, appears to be relatively insensitive to the effects of preload alterations (11,12,14). However, its clinical implications and reliability as an indicator of LV diastolic function remain to be elucidated. We therefore investigated the relation between peak negative MVG and LV diastolic performance in patients with HCM.

METHODS

Study group. We studied 36 patients (34 men, 2 women) with newly diagnosed nonfamilial, nonobstructive HCM, with a mean age of 54 ± 11 years (range 35 to 72 years). The diagnosis was based on conventional echocardiographic demonstration of a nondilated, hypertrophied left ventricle without evidence of LV outflow tract obstruction and in the absence of other cardiac or systemic diseases that might lead to LV hypertrophy (15); it was confirmed by cardiac catheterization, angiography, and endomyocardial biopsy. All patients were in normal sinus rhythm and had a normal LV ejection fraction as revealed by left ventriculography. The control group consisted of 47 subjects (40 men, 7 women; mean age 39 ± 18 years; range 22 to 78 years) with normal electrocardiograms (ECG) or nonspecific ST-T changes and with normal conventional echocardiograms. We excluded patients with localized asynergy revealed by conventional two-dimensional echocardiograms or left ventriculograms. We excluded patients with localized asynergy revealed by conventional two-dimensional echocardiograms or left ventriculograms.

In a subset of 26 age-matched controls (24 men, 2 women; mean age 52 ± 14 years; range 26 to 78 years), cardiac catheterization was performed for evaluation of atypical chest pain or discomfort. These individuals had normal left ventriculograms and did not have valvular heart disease or a >50% narrowing of the coronary arteries as revealed by coronary angiography. Therefore, they were classified as normal subjects.

All drugs were discontinued at least four days before subject evaluation. The study was approved by the appro-

Abbreviations and Acronyms

dP/dt = first derivative of left ventricular pressure  
ECG = electrocardiogram  
HCM = hypertrophic cardiomyopathy  
LV = left ventricular  
LVEDP = left ventricular end-diastolic pressure  
MVG = myocardial velocity gradient  
PAWP = pulmonary artery wedge pressure  
tau = time constant of left ventricular pressure decay  
TDI = tissue Doppler imaging  
T\^{D} = tau determined by the derivative method  
T\^{1/2} = tau determined by the pressure half-time method

priate institutional review committee. Subjects were informed in detail of the purpose and methods of the study, and they provided written informed consent.

Cardiac catheterization. Subjects received 5 mg of oral diazepam before catheterization. A 6F pigtail angiographic high-fidelity micromanometer-tipped catheter (model SPC-464D, Millar Instruments, Houston, Texas) was advanced into the left ventricle through the right brachial artery for measurement of LV pressure. The micromanometer pressure was matched to the pressure of the fluid-filled lumen. A 21-gauge catheter was placed in the left brachial artery for arterial pressure measurements. To measure pulmonary artery wedge pressure (PAWP) and cardiac output, we positioned a 7F triple-lumen thermodilution Swan-Ganz catheter (Baxter Healthcare, Deerfield, Illinois) in the pulmonary artery through the right brachial vein. A PAWP transducer (model 746, Siemens Medical Systems, Solna, Sweden) was placed at the zero reference point at the midchest level. The ECGs were recorded with the Mason Liker modification of the standard 12-lead ECG. We recorded micromanometer pressure signals and ECGs simultaneously and continuously with a multichannel recorder (MR-40, TEAC, Tokyo, Japan). All subjects underwent selective coronary angiography, left ventriculography, and endomyocardial biopsy.

Standard echocardiography and TDI. Standard echocardiography, including measurement of transmural flow velocity indexes, was performed with a Powervision 8000 ultrasonoscope (Toshiba Medical Systems, Tochigi, Japan), and images were recorded on 0.5-inch videotape with VHS recorders. Septal and posterior wall thickness and LV end-diastolic and –systolic dimensions were measured, and LV fractional shortening was determined from LV M-mode recordings. Echocardiographic LV mass was calculated by the area-length method as recommended by the American Society of Echocardiography (16). The LV mass index was calculated by dividing the LV mass by body surface area. Peak early and late transmural filling velocities, the deceleration time of peak early velocity, and the isovolumic relaxation time were measured from mitral inflow velocities. On completion of the standard echocardiographic measurements, TDI was performed to obtain peak negative MVG.

Two examiners who were unaware of the clinical status of the subject performed the echocardiographic analysis. Representative recordings of the ECG, LV pressure, and dP/dt together with M-mode echocardiograms and TDI for an HCM patient are shown in Figure 1.

Data analysis. The LV pressure signals were digitized and analyzed with software developed in our laboratory and a 32-bit microcomputer system (PC-9821-ST20, NEC, Tokyo, Japan) (17). We calculated the peak positive dP/dt as an index of contractility. To evaluate LV isovolumic relaxation, we calculated τ in two different ways. The first approach was based on the direct measurement of the pressure half-time (T\^{1/2}) as described by Mirsky (18); this parameter was computed for each acquisition as the time
required for the pressure at the time of peak negative dP/dt to decrease by 50% of its value. The second approach was based on that described by Raff and Glantz (19), in which tau (\(T_D\)) is determined from the negative inverse slope of the relation between LV pressure and dP/dt. Pressure data from that at peak negative dP/dt to a value 5 mm Hg greater than the previous LVEDP were used for this calculation.

**Calculation of MVG in M-mode TDI.** The angle of interrogation of the M-mode beam was aligned to be perpendicular to the LV posterior wall in long-axis view.
Freeze-frame images of conventional gray-scale and the corresponding color-coded velocity mapping images in a bidirectional red-and-blue mode were then downloaded to a magneto-optic disk and transferred to an IBM-compatible computer for off-line analysis of myocardial velocities and gradient by custom-made software (PowerView, Toshiba Medical Systems, Tochigi, Japan), as previously described (11,14,20). Velocity ranges were adjusted to maximize the sensitivity for low-velocity values while avoiding saturation of the highest velocity during diastole. Use of M-mode TDI yielded a temporal resolution of as high as 4 ms.

With M-mode TDI, endocardial and epicardial velocities were calculated within the posterior wall. The MVG was defined as the slope of a linear regression of the myocardial velocity profile along each M-mode scan line throughout the myocardium, which reflects the dynamics of wall thickening and thinning (8,9). The MVG calculations were repeated at each time point throughout the cardiac cycle. Peak negative MVG in early diastole was subsequently determined and was averaged for three consecutive beats.

Statistical analysis. Data are presented as means ± SD. Normality was evaluated by normal distribution plots and histograms for the variable. Characteristics and hemodynamic variables were compared between groups by the Student unpaired two-tailed t test. The relations between peak negative MVG and hemodynamic variables were analyzed with the use of the Pearson correlation coefficient. A p value of <0.05 was considered statistically significant. The cutoff value of the peak negative MVG for differentiation between HCM patients and 47 normal individuals was determined with the use of a receiver-operating characteristic curve. The sensitivity [true positive/(true positive + false negative)], specificity [true negative/(true negative + false positive)], and predictive accuracy [(true positive + true negative)/total group] were determined and expressed as percentages.

RESULTS

Age and gender distribution did not differ between patients with HCM and the subset of controls subjected to cardiac catheterization (Table 1). Variables of noninvasive and invasive parameters were normally distributed. The peak negative MVG was significantly smaller in HCM patients than in the entire control group (2.32 ± 0.52/s vs. 4.82 ± 1.15/s, p < 0.0001) (Fig. 2) or in controls subjected to catheterization (Table 1). Neither LV fractional shortening, transmitral flow indexes, the deceleration time of peak early velocity, nor the isovolumic relaxation time differed significantly between patients with HCM and controls. The cutoff value of normal peak negative MVG was 3.2/s and was used to differentiate between 47 normal individuals and HCM patients, yielding sensitivity, specificity, and predictive accuracy values of 97.2% (35 of 36 subjects), 95.7% (45 of 47 subjects), and 96.4% (80 of 83 subjects), respectively.

Simultaneous recording of LV pressure and LV dP/dt by cardiac catheterization together with the M-mode TDI revealed that the period of peak negative MVG corresponded to the initial phase of rapid filling after isovolumic relaxation (Fig. 1); that is, the peak negative MVG coincided with the process of auxotonic relaxation during LV filling with variable LV pressure. Heart rate, cardiac index, and LV peak positive dP/dt did not differ significantly between HCM patients and control subjects (Table 1). The LVEDP, PAWP, and tau (both $T_{1/2}$ and $T_D$) were significantly increased in HCM patients compared with controls.

Correlation analysis with the Pearson correlation coefficient revealed that the peak negative MVG was inversely correlated with LVEDP ($r = -0.75, p < 0.0001$ [Fig. 3]), PAWP ($r = -0.70, p < 0.0001$ [Fig. 4]), and tau ($T_{1/2}, r = -0.58, p < 0.0001$ [Fig. 5]; $T_D, r = -0.64, p < 0.0001$). Evaluation of data from HCM patients alone revealed that the peak negative MVG was also significantly correlated with LVEDP ($r = -0.52, p = 0.001$), PAWP ($r = -0.59, p < 0.0001$), and tau ($T_{1/2}, r = -0.47, p = 0.003$; $T_D, r = -0.39, p = 0.02$). None of the other echocardiographic indexes, including conventional Doppler indexes, correlated significantly with hemodynamic variables.

### Table 1. Characteristics, Hemodynamic Variables, Conventional Echocardiographic Data Including Doppler Indexes, and TDI Indexes of HCM Patients and Controls Subjected to Cardiac Catheterization

<table>
<thead>
<tr>
<th>Parameter</th>
<th>HCM (n = 36)</th>
<th>Controls (n = 26)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>54 ± 11</td>
<td>52 ± 14</td>
<td>NS</td>
</tr>
<tr>
<td>Male subjects (%)</td>
<td>94.4</td>
<td>92.3</td>
<td>NS</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.73 ± 0.22</td>
<td>1.72 ± 0.18</td>
<td>NS</td>
</tr>
<tr>
<td>LVdD (mm)</td>
<td>48.2 ± 5.7</td>
<td>46.8 ± 4.8</td>
<td>NS</td>
</tr>
<tr>
<td>LVDs (mm)</td>
<td>30.4 ± 6.7</td>
<td>28.9 ± 4.5</td>
<td>NS</td>
</tr>
<tr>
<td>IVST (mm)</td>
<td>16.6 ± 3.6</td>
<td>10.1 ± 1.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>PWT (mm)</td>
<td>11.1 ± 1.8</td>
<td>9.7 ± 0.9</td>
<td>0.01</td>
</tr>
<tr>
<td>LVMi (g/m²)</td>
<td>198.6 ± 49.9</td>
<td>119.6 ± 29.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>FS (%)</td>
<td>37.4 ± 7.3</td>
<td>38.5 ± 5.6</td>
<td>NS</td>
</tr>
<tr>
<td>Peak E velocity (cm/s)</td>
<td>66.3 ± 14.3</td>
<td>74.3 ± 17.5</td>
<td>NS</td>
</tr>
<tr>
<td>Peak A velocity (cm/s)</td>
<td>74.0 ± 16.1</td>
<td>68.7 ± 13.9</td>
<td>NS</td>
</tr>
<tr>
<td>E/A velocity ratio</td>
<td>0.9 ± 0.35</td>
<td>1.1 ± 0.38</td>
<td>NS</td>
</tr>
<tr>
<td>IRT (ms)</td>
<td>119.0 ± 14.0</td>
<td>91.0 ± 22.3</td>
<td>NS</td>
</tr>
<tr>
<td>dP/dt (mm Hg/s)</td>
<td>1226.8 ± 30.7</td>
<td>1807.0 ± 29.8</td>
<td>NS</td>
</tr>
<tr>
<td>Peak negative MVG (/s)</td>
<td>2.2 ± 0.52</td>
<td>4.8 ± 1.22</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>66 ± 8.6</td>
<td>64 ± 11.4</td>
<td>NS</td>
</tr>
<tr>
<td>CI (l/min per m²)</td>
<td>3.0 ± 0.5</td>
<td>2.8 ± 0.6</td>
<td>NS</td>
</tr>
<tr>
<td>PAWP (mm Hg)</td>
<td>141.1 ± 4.3</td>
<td>73.2 ± 2.8</td>
<td>&lt;0.0001</td>
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<td>LVEDP (mm Hg)</td>
<td>19.6 ± 6.1</td>
<td>6.5 ± 1.7</td>
<td>&lt;0.0001</td>
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<tr>
<td>Peak + dP/dt (mm Hg/s)</td>
<td>1705 ± 438</td>
<td>1912 ± 436</td>
<td>NS</td>
</tr>
<tr>
<td>Peak − dP/dt (mm Hg/s)</td>
<td>1637 ± 457</td>
<td>1890 ± 302</td>
<td>NS</td>
</tr>
<tr>
<td>$T_{1/2}$ (ms)</td>
<td>44.0 ± 6.7</td>
<td>32.1 ± 5.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>$T_D$ (ms)</td>
<td>57.6 ± 14.9</td>
<td>37.1 ± 5.7</td>
<td>&lt;0.0001</td>
</tr>
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</table>

Data are means ± SD.

BSA = body surface area; CI = cardiac index; DcT = deceleration time of E velocity; dP/dt = first derivative of left ventricular pressure; FS = fractional shortening; IRT = isovolumic relaxation time; IWT = thickness of interventricular septum; LVDd = left ventricular end-diastolic internal dimension; LVDs = left ventricular end-systolic internal dimension; LVEDP = left ventricular end-diastolic pressure; LVMi = left ventricular mass index; MVG = myocardial velocity gradient; PAWP = pulmonary artery wedge pressure; Peak A velocity = peak late transmitral filling velocity; Peak E velocity = peak early transmitral filling velocity; PWT = thickness of left ventricular posterior wall; $T_{1/2}$ and $T_D$ = tau calculated by direct pressure half-time method and derivative method, respectively.
DISCUSSION

We have shown that (1) the period of peak negative MVG corresponds to the process of auxotonic relaxation during the initial phase of rapid filling associated with variable LV pressure that follows isovolumic relaxation; (2) the peak negative MVG is inversely correlated with LVEDP, PAWP, and tau; (3) the peak negative MVG is significantly reduced in patients with HCM compared with controls; and (4) the cutoff value for normal peak negative MVG was 3.2/s. Determination of peak negative MVG by the noninvasive technique of TDI thus provides an estimate of the increase in LVEDP as well as the prolongation of tau in HCM patients.

Figure 2. Comparison of peak negative myocardial velocity gradient (MVG) between hypertrophic cardiomyopathy (HCM) patients and control subjects.

Figure 3. Correlation between peak negative myocardial velocity gradient (MVG) and left ventricular end-diastolic pressure (LVEDP) for all individuals subjected to cardiac catheterization (solid line; closed and open circles represent hypertrophic cardiomyopathy (HCM) patients and controls, respectively) and for HCM patients alone (dotted line).
Peak negative MVG during the cardiac cycle. Measurements of diastolic function can be divided into those that reflect the process of active relaxation and those that reflect passive stiffness. This division is somewhat arbitrary, however, given that structures and processes affecting relaxation can also result in measurable abnormalities in stiffness (21). At the chamber level, the diastolic process results in a reduction in LV pressure at a constant volume (isovolumic relaxation), which is followed by LV filling at variable LV pressures (auxotonic relaxation). Consequently, it is essen-

**Figure 4.** Correlation between peak negative myocardial velocity gradient (MVG) and pulmonary artery wedge pressure (PAWP) for all individuals subjected to cardiac catheterization (solid line; closed and open circles represent hypertrophic cardiomyopathy (HCM) patients and controls, respectively) and for HCM patients alone (dotted line).

**Figure 5.** Correlation between peak negative myocardial velocity gradient (MVG) and tau determined by the derivative method ($T_{1/2}$) for all individuals subjected to cardiac catheterization (solid line; closed and open circles represent hypertrophic cardiomyopathy (HCM) patients and controls, respectively) and for HCM patients alone (dotted line).
tial to quantify LV performance, including both active relaxation and passive stiffness, in order to assess diastolic dysfunction accurately. We now show that the period of peak negative MVG corresponds to auxotonic relaxation, the initial phase of rapid filling with variable LV pressure. The peak negative MVG is thus a novel indicator of auxotonic relaxation and may prove useful as a predictor of diastolic dysfunction characterized by increased stiffness and prolonged active relaxation.

Although LV filling does not occur during isovolumic relaxation, the process that determines the rate of decrease in pressure during isovolumic relaxation influences ventricular filling after opening of the mitral valve (22,23). For the first 30 to 40 ms after mitral valve opening, relaxation of LV wall tension causes the LV pressure to fall (24), thereby generating a pressure gradient and resulting in rapid filling. After diastasis, atrial contraction increases the atrial pressure and propels blood into the left ventricle. Therefore, LV isovolumic relaxation influences the period of auxotonic relaxation, during which the peak negative MVG occurs; this period also influences the entire LV filling performance.

Both tau and peak negative dP/dt derived from invasive techniques are well established as important clinical and research tools for evaluation of LV relaxation abnormalities (2,19,22). The peak negative dP/dt is affected by loading conditions. Although tau is not significantly influenced by preload alterations within a physiologic range (25), it is estimated from the slope of the natural logarithm of the pressure–time relation, reflecting the descending curve of LV dP/dt, and corresponds to only the first period of the isovolumic phase. The peak negative MVG reflects the auxotonic relaxation phase, which is affected by both LV active relaxation and passive stiffness. Our findings are consistent with previous observations showing that the early velocity of the mitral annulus obtained by TDI reflects PAWP, behaves as a preload-independent index of LV relaxation, and may be used to estimate LV filling pressure (26,27).

**Peak negative MVG in HCM patients.** The peak negative MVG was significantly smaller in patients with HCM than in control subjects, even though systolic performance was similar in both groups. The cutoff value of the normal peak negative MVG was 3.2/s. The LVEDP and PAWP were significantly higher and tau was significantly prolonged in HCM patients compared with controls. The LVEDP, PAWP, and tau are widely used as indexes of diastolic function (2,17,18,28) and were shown to be inversely correlated with peak negative MVG in the present study. The MVG is derived from the regression line for the transmural velocity profile rather than from the endocardial and epicardial point velocity difference (8,9). The pathologic features of HCM, which include fibrosis and disarray of the myocardium resulting in diminished ventricular compliance (1,15), impaired relaxation, and increased LVEDP and PAWP, are responsible for the deterioration in the dynamics of myocardial thickening and thinning. Hence, the reduction in peak negative MVG may provide insight into the pathogenesis of HCM.

**Clinical implications.** Diastolic heart failure accounts for 30% to 50% of all cases of heart failure with preserved LV systolic function, and it has become recognized as a separate clinical entity (29–31). It has thus become important to establish precise indexes for assessment of diastolic function. Peak negative MVG has previously been suggested as an indicator of LV relaxation (11–13) that is relatively independent of changes in preload (11,12,14). However, we have shown that the peak negative MVG provides information related to LV diastolic performance, including both active relaxation and passive stiffness, and thus may prove of use as a noninvasive indicator of global LV diastolic function. Serial measurements of peak negative MVG should prove valuable in the follow-up of patients with progressive diastolic dysfunction.

**Study limitations.** Both the pattern and the extent of LV hypertrophy vary greatly among patients with HCM, although abnormalities of global diastolic filling are largely independent of the extent and distribution of myocardial hypertrophy (32). It remains unclear, however, whether diastolic filling varies among different regions of the left ventricle and whether it is influenced by the thickness of the septum (33). In addition, wall motion abnormalities such as asynchrony that might result from ischemic events may influence the accuracy of the determination of peak negative MVG. For patients either with regional asynergy due to ischemic events or with asymmetric hypertrophy, the evaluation of global LV diastolic abnormalities solely from the MVG of the posterior wall will provide insufficient information. Development of angle-independent, two-dimensional TDI that would allow both examination of all segments of the LV wall and comparison of regional diastolic function is awaited in the near future.

We required a sufficient number of control subjects to determine the cutoff value of peak negative MVG for differentiation between HCM patients and normal individuals with a receiver operating characteristic curve. Given that it was not ethically justified to perform invasive measurements with healthy subjects, in addition to the 26 controls who underwent cardiac catheterization, we included 21 control subjects who did not undergo this procedure in the determination of the cutoff value.

Mitral annular velocity correlates with left atrial pressure and mean PAWP, and this can be used to estimate LV filling pressure (26,27). Also, LVEDP can be evaluated from the relation of mitral A-wave duration to pulmonary vein A-wave duration (34,35). Further studies are required to compare the reliability of estimation of LVEDP or PAWP from peak negative MVG and from other noninvasive indexes, not only in patients with HCM but also more generally in individuals with diastolic dysfunction.

**Conclusions.** Our results suggest that the peak negative MVG is an indicator of global LV diastolic function, reflecting the process of auxotonic relaxation, and provides...
information about both passive stiffness and active relaxation. It should therefore prove to be a useful index in the follow-up of patients with progressive diastolic dysfunction secondary to HCM.

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