Use of Color Kinesis for Evaluation of Left Ventricular Filling in Patients With Dilated Cardiomyopathy and Mitral Regurgitation

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Objectives. We tested the feasibility of using analysis of color kinesis images to objectively assess global and regional left ventricular (LV) diastolic function in patients with dilated cardiomyopathy (DCM). In addition, the ability of this technique to track drug-induced changes on LV diastolic properties was studied.

Background. Diastolic dysfunction contributes to symptomatology in patients with DCM. The assessment of LV diastolic function using conventional Doppler echocardiography is indirect and is confounded by multiple variables. Moreover, the noninvasive evaluation of regional diastolic properties is difficult. In contrast, color kinesis directly tracks and color-encodes regional diastolic endocardial motion.

Methods. We studied 24 patients with DCM and mitral regurgitation (MR) and 24 age-matched normal subjects. Transmitral and pulmonary vein flow velocities were measured using pulsed Doppler echocardiography. Diastolic color kinesis images were obtained in patients with DCM and in normal subjects. Compared with Doppler indexes, color kinesis showed significant differences between patients with DCM and normal subjects. Compared with Doppler indexes, color kinesis was less confounded by MR and was capable of differentiating between drug-induced lusitropic and vasodilator effects. Diastolic asynchrony was increased in patients with DCM and severe MR.

Results. Color kinesis indexes of global diastolic function showed significant differences between patients with DCM and normal subjects. Compared with Doppler indexes, color kinesis was less confounded by MR and was capable of differentiating between drug-induced lusitropic and vasodilator effects. Diastolic asynchrony was increased in patients with DCM and severe MR.

Conclusions. Quantitative analysis of global and regional LV diastolic function in patients with DCM using color kinesis is feasible.

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Seven additional patients were screened and excluded because their two-dimensional image quality allowed visualization and tracking of <75% of the endocardial boundary. The etiologies for DCM were idiopathic (n = 12), ischemic (n = 8), postpartum (n = 3) and postmyocarditis (n = 1). Five patients with DCM had left bundle branch block (LBBB). An additional group of seven patients with DCM was enrolled according to the same inclusion criteria to evaluate the effect of drugs, as is detailed in the description of protocol 3.

Twenty-four normal subjects (10 men and 14 women, mean age 54 ± 14 years [range 33 to 88]) were used as the age-matched control group. Inclusion criteria for this group included 1) normal two-dimensional echocardiographic study without evidence of regional wall motion abnormalities or valvular heart disease; 2) normal LV mass index; 3) normal sinus rhythm without conduction abnormalities; and 4) normal mitral flow velocity, ratio of early to late diastolic mitral flow velocity (E/A ratio) and deceleration time, according to age and gender (14).

Data acquisition. Two-dimensional and M-mode echocardiography. Images were acquired using either a 2.5- or 3.5-MHz transducer (SONOS 2500, Hewlett-Packard). Parasternal mid-papillary short-axis and apical four-chamber views of the LV were obtained in the left lateral decubitus position and recorded on videotape for off-line measurements of LV mass index. Two-dimensional targeted M-mode imaging of the LV was performed in the parasternal short-axis view at the mid-papillary muscle level. Tracings were recorded on paper at a speed of 50 mm/s to measure chamber dimensions. In addition, MR in patients with DCM was quantified by measuring the atrial regurgitation area using Doppler color flow imaging (15).

Pulsed Wave Doppler echocardiography. Initially, the sample volume was placed at the junction between the anterior mitral leaflet and the aortic root to obtain the isovolumetric relaxation time (IVRT). Subsequently, in the apical four-chamber view, mitral inflows and pulmonary vein flow were recorded as previously described (9). All Doppler tracings were acquired during passive end-expiration at 100 mm/s, with the filter set at the lowest level possible, and recorded on videotape. Electrocardiograms (ECGs) and respiration signals were also recorded.

Color kinesis. Diastolic color kinesis images were obtained in each subject from the LV mid-papillary short-axis and apical four-chamber views. Initially, the acoustic quantification system was activated and the gain controls (total and lateral gains, time gain compensation) adjusted to optimize tracking of the endocardial border (16). Color kinesis was then activated to color-encode diastolic LV endocardial motion. Accuracy of endocardial tracking was confirmed by toggling on and off the color overlays. Fine adjustments of regional gain setting were then performed to optimize endocardial tracking of specific segments.

Time settings were manually adjusted when necessary to ensure that color-encoding started with the first video frame in which outward endocardial motion was noted. Subsequently, the duration of the diastolic sequence was set to its maximal value (19 frames) to ensure color-encoding of the entire LV filling period. Three nonconsecutive end-diastolic color kinesis images were acquired in each view and stored on optical disk for off-line analysis.

Data analysis. LV mass index measurements. LV mass was measured using the two-dimensional area–length method with the software incorporated in the ultrasound system. End-diastolic (R wave peak) short-axis epicardial and endocardial LV borders were manually traced using electronic calipers. Long-axis length was measured from the apical four-chamber view as the distance between the mid-mitral annulus plane and the tip of the apical endocardium.

M-mode echocardiography. Using the conventional leading edge technique, measurements of both end-diastolic and end-systolic LV cavity diameters were performed and shortening fraction calculated.

Doppler echocardiography. Measurements were performed off-line using the Doppler analysis package incorporated in the echocardiographic system. All values were obtained as the mean of three nonconsecutive beats. IVRT was measured as the interval between the aortic valve closure click and the onset of the mitral valve inflow. When atrial contraction occurred before mitral flow velocity reached the zero level, the deceleration phase was linearly extrapolated to the baseline. Only cardiac cycles with linear deceleration and clearly defined early and late diastolic velocity peaks were used for measurements. Peak velocities during rapid filling (E wave) and atrial contraction (A wave) were measured, and the E/A ratio calculated.

Pulmonary vein inflow velocities were measured at end-expiration. The ratio of peak forward flow velocity from ventricular systole to diastole (S/D ratio) was calculated. In addition, the maximal velocity of the diastolic pulmonary vein flow reversal at atrial contraction (pv-a) was measured, and the mitral A wave/pv-a ratio calculated.

Color kinesis images. End-diastolic color-encoded images were automatically divided into six segments using custom software (7,8). Briefly, the segmentation originated from the automatically determined end-systolic centroid of the LV cavity and was based on a few easily identifiable anatomic

Abbreviations and Acronyms
A wave  = peak velocity of mitral inflow during atrial contraction
DCM  = dilated cardiomyopathy
E/A  = ratio of early to late transmitral flow velocity
E wave  = peak velocity of mitral inflow during rapid left ventricular filling
IVRT  = isovolumetric relaxation time
LV  = left ventricle, ventricular
LBBB  = left bundle branch block
MR  = mitral regurgitation
PFR  = peak (left ventricular) filling rate
pv-a  = maximal velocity of diastolic pulmonary vein flow reversal at atrial contraction
S/D  = ratio of peak forward pulmonary vein flow velocity from ventricular systole to diastole
To obtain 20 values of segmental filling fraction in beats/s.

To compensate for differences in specific segment. This display allowed easy identification of the filling rate as a function of time. These data were displayed as a stacked time-histogram, wherein each layer represents one percent segmental end-diastolic area. This variable was divided by instantaneous heart rate peak (LV) filling rate (PFR). To compensate for differences in heart rate, PFR was normalized by instantaneous heart rate.

To evaluate the temporal filling patterns on a regional basis, the filling fraction was integrated with respect to time and normalized to 100% in each segment. This normalization, together with the aforementioned normalization of the time axis, resulted in a display wherein each curve reached 100% of diastolic endocardial motion at 100% filling time. These data were used to generate regional time curves, which reflected the percent diastolic filling fraction of each segment at any specific percent LV filling time. Percent endocardial expansion at each 25%, 50% and 75% of LV filling time was averaged for all segments in the parasternal short-axis view, and the standard deviation of the mean was used as an index of diastolic asynchrony at each of these particular times of LV filling.

The study consisted of three different protocols. The aim of Protocol 1 was to compare the clinical utility of color kinesis and Doppler echocardiography for the assessment of global diastolic function in patients with DCM in the presence of MR. Accordingly, mitral and pulmonary venous Doppler studies as well as diastolic color kinesis images were acquired and analyzed as described earlier in patients with DCM and in normal subjects.

Protocol 2 was designed to test the feasibility of using the aforementioned segmental analysis of color kinesis images to objectively assess regional diastolic function in patients with DCM and in normal subjects. Initially we tested the ability of color kinesis to identify regional diastolic asynchrony by comparing the index of asynchrony at 25%, 50% and 75% of LV filling time obtained in a group of patients with DCM and LBBB. The findings in these patients were compared with those in patients with DCM and normal conduction. Subsequently, to evaluate the effects of MR on color kinesis indexes of diastolic asynchrony, the 19 patients with DCM and normal conduction were subclassified into two subgroups according to the severity of MR: moderate MR (regurgitation area <4 cm², n = 11) and severe MR (regurgitation area ≥4 cm², n = 8) (15).

In Protocol 3, we compared the ability of Doppler echocardiography and color kinesis to detect the differential effects of two pharmacologic agents, nitroprusside and dobutamine, on LV diastolic properties. Seven patients with DCM (5 men and 2 women, age 57 ± 8 years) were enrolled. Data were acquired under 1) control conditions; 2) infusion of nitroprusside (1.5 μg/kg body weight per min), a nonspecific venous and arterial vasodilator; and 3) infusion of dobutamine (7.5 μg/kg per min), a beta₁-adrenoceptor agonist with positive inotropic and lusitropic effects. Dobutamine infusion was initiated 20 min after termination of the nitroprusside infusion.

Statistical analysis. For each subject, data obtained from three end-diastolic color kinesis images were averaged. Doppler values were obtained as the mean of three nonconsecutive beats. Analysis of variance was used for comparisons between patients and normal subjects and applied with correction for even 5% increments of the LV filling time. Thus, irrespective of heart rate, the LV filling time was normalized to 100% in each subject. In addition, the mean time of LV filling was calculated and normalized by the RR interval (7,8).

To facilitate intersubject comparisons, we used linear interpolation to obtain 20 values of segmental filling fraction in beats/s.
Table 1. Echocardiographic Data

<table>
<thead>
<tr>
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<th>Normal Subjects (n = 24)</th>
<th>Patients With DCM (n = 24)</th>
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<tbody>
<tr>
<td>M-mode and two-dimensional echocardiography</td>
<td></td>
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<tr>
<td>Fractional shortening (%)</td>
<td>43 ± 6</td>
<td>16 ± 5*</td>
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<tr>
<td>LV mass index (g/m²)</td>
<td>66 ± 13</td>
<td>124 ± 42*</td>
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<tr>
<td>Doppler echocardiography</td>
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<tr>
<td>E wave (cm/s)</td>
<td>79 ± 12</td>
<td>85 ± 37</td>
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<tr>
<td>A wave (cm/s)</td>
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<td>86 ± 32</td>
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<td>E/A</td>
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<tr>
<td>DT/RR</td>
<td>221 ± 41</td>
<td>250 ± 93</td>
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<td>IVRT/RR</td>
<td>78 ± 22</td>
<td>126 ± 29*</td>
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<tr>
<td>S/D</td>
<td>1.3 ± 0.3</td>
<td>0.9 ± 0.5*</td>
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<tr>
<td>Color kinesis</td>
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<tr>
<td>Filling fraction (%EDA)</td>
<td>53 ± 7</td>
<td>27 ± 8*</td>
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<tr>
<td>PFR/HR (EDA/beat)</td>
<td>2.4 ± 0.5</td>
<td>1.2 ± 0.4*</td>
</tr>
<tr>
<td>MFT/RR</td>
<td>244 ± 29</td>
<td>210 ± 26*</td>
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</table>

*p < 0.05 compared with normal subjects. Data are presented as mean value ± SD. DCM = dilated cardiomyopathy; DT = deceleration time; E/A = ratio of early to late transmitral flow velocities; EDA = end-diastolic area; HR = heart rate (s); IVRT = isovolumetric relaxation time; LV = left ventricular; MFT = mean filling time; PFR = peak filling rate; RR = RR interval (s); SD = ratio between peak velocities of forward pulmonary vein flow during systole (S wave) and diastole (D wave).

Results

Protocol 1. A summary of the results of the two-dimensional echocardiographic, Doppler echocardiographic and color kinesis measurements of LV diastolic function in normal subjects and in patients with DCM is presented in Table 1. As expected, in patients with DCM, LV fractional shortening was significantly reduced, LV mass index was higher and heart rate was elevated (82 ± 10 vs. 68 ± 8 beats/min, p < 0.0001), as compared with normal subjects. The mean MR area in patients with DCM was 4.7 ± 2.6 cm² according Doppler color flow imaging.

Analysis of transmitral filling dynamics showed no significant intergroup differences in either E and A waves, E/A ratio or deceleration time. The IVRT was significantly prolonged in patients with DCM as compared with normal subjects (Table 1). To compensate for differences in heart rate, we also analyzed IVRT and deceleration time normalized by the RR interval and found similar results. Analysis of pulmonary vein flow showed a reduced S/D ratio in patients with DCM as compared with normal subjects. No significant changes of pv-a were found; however, the mitral A wave/pv-a ratio was significantly increased in patients with DCM as compared with normal subjects (Table 1).

Figure 1 shows an example of end-diastolic color kinesis images acquired in a patient with DCM and in a normal subject. Significant differences in all color kinesis indexes of global LV diastolic function were found between patients with DCM and age-matched control subjects. Both filling fraction and PFR, as well as the mean filling time, were significantly reduced in patients with DCM (Table 1). In this study, the best three predictors of diastolic dysfunction, according to the stepwise forward regression analysis, were 1) PFR; 2) mean filling time; and 3) IVRT, with a combined r² of 0.82.

Protocol 2. Segmental analysis of color kinesis images allowed quantification of the magnitude and timing of regional diastolic endocardial motion in patients with DCM and in normal subjects. The stacked histograms shown in Figure 2 demonstrate that the decrease in global filling fraction in patients with DCM, as noted in Protocol 1, was a result of a diffuse decrease in regional diastolic endocardial motion. Figure 3 presents filling rate time-histograms for all endocardial segments in the short-axis view. These time histograms show that the decrease in global LV PFR is also a result of a decrease in regional peak filling rates in all individual segments.

Figure 4 (top) shows an example of regional time curves obtained in a normal subject, which depict uniform regional endocardial motion with minor intersegmental heterogeneity. The heterogeneity was highest during early filling and diminished as filling progressed (Table 2). Figure 4 (middle) shows similar data obtained in a patient with DCM and LBBB. The increased asynchrony noted in this patient was due to delayed endocardial motion in the LV lateral wall, whereas the remaining segments showed synchronous diastolic endocardial motion similar to that seen in the normal subject (Fig. 4, top). Similar patterns of regional diastolic endocardial motion were noted in three of five patients with LBBB. Consequently, the LBBB subgroup had a significantly increased index of asynchrony as compared with the group of normal subjects during the entire LV filling period (Table 2). Figure 4 (bottom) shows an example of regional filling curves obtained in a patient with DCM and severe MR, which also demonstrate increased diastolic asynchrony. However, in contrast to the patient with LBBB, all segments moved asynchronously during LV filling. As a group, patients with DCM and severe MR had a tendency toward increased diastolic asynchrony as compared with control subjects and patients with moderate MR, who were similar to normal subjects (Table 2).

Protocol 3. The results of this protocol are shown in Table 3. Neither nitroprusside nor dobutamine infusion resulted in
Figure 2. Stacked histograms of regional filling fraction (FF) in percent regional end-diastolic area (REDA) obtained from end-diastolic color kinesis images in normal subjects (NL, left) and patients with DCM (right) in the parasternal short-axis (SAX, top) and apical four-chamber (A4C, bottom) views. Different colors correspond to those used in color kinesis images and represent incremental area change in consecutive video frames (see color-encoding scheme at the bottom). Compared with normal subjects, patients with DCM had a significantly lower filling fraction in all endocardial segments. Data are expressed as the mean value ± SD (gray band), ant = anterior; asp = anteroseptal; sp = septal; inf = inferior; pst = posterior; lat = lateral; b-lt = basal lateral; m-lt = mid-lateral; a-lt = apical lateral; a-sp = apical septal; m-sp = mid-septal; b-sp = basal septal.

significant variations in mean heart rate (87 ± 6 beats/min at baseline, 91 ± 6 beats/min with nitroprusside and 89 ± 11 beats/min with dobutamine). The administration of nitroprusside and dobutamine resulted in similar trends toward normalization in all three Doppler variables—namely, a reduction in the E/A ratio and IVRT and an increase in the S/D ratio (Fig. 5).

With both drugs, PFR increased toward its normal value (Fig. 5); this normalization trend was more pronounced with dobutamine. In contrast, mean filling time was increased with nitroprusside and reduced with dobutamine (Fig. 5). These opposing effects were statistically significant. The analysis of regional diastolic wall motion showed a nonsignificant reduction in the early asynchrony index measured at 25% of LV filling time, from 11.3 ± 2.9 at baseline to 9.5 ± 3.8 with dobutamine and 9.5 ± 3.1 with nitroprusside.

Discussion

Reduced functional capacity is a cardinal symptom of congestive heart failure. The impairment in functional capacity, however, correlates poorly with the severity of LV systolic dysfunction (17–23). Diastolic dysfunction in patients with DCM has been previously demonstrated using both invasive and noninvasive methods (6,12,24–26). Most noninvasive studies were based on Doppler echocardiographic indexes, which indirectly assess LV diastolic function by measuring instantaneous blood flow velocities. These indexes are known to be confounded by multiple factors, such as loading conditions, age and heart rate (27). Accordingly, new echocardiographic methods, such as acoustic quantification, color flow propagation and Doppler tissue imaging, have been proposed to obtain objective assessment of LV diastolic function (11,28–33). However, most of these techniques allow either a global or limited regional analysis of LV diastolic function.

In contrast, color kinesis is a new echocardiographic technique that allows direct quantification of the magnitude and timing of regional LV endocardial motion by tracking and color-encoding pixel transitions from tissue to blood during LV filling. Although the accuracy of this new technique requires further validation, in this study, color kinesis images were analyzed to test the feasibility of quantitative assessment of global and regional LV diastolic properties in patients with DCM. For comparison, indexes of diastolic function derived from transmitral and pulmonary vein pulsed Doppler echocardiography were also obtained.

Doppler evaluation of global diastolic function. Our results were in agreement with previous studies (6,34) suggesting that MR masks LV filling abnormalities analyzed by mitral inflow Doppler in patients with DCM. Specifically, transmitral flow velocities in these patients were not different from those of age-matched normal subjects. The confounding effects of MR on the assessment of LV diastolic function may be explained by the findings described by Appleton et al. (5) and
Choong et al. (35). In these studies, mitral Doppler indexes were affected by altered loading condition, whereas the invasively acquired IVRT constant, which is considered to be the best index of LV relaxation characteristics, remained relatively unaltered.

Pulmonary vein filling of the left atrium evaluated by pulsed Doppler echocardiography has been recently added as a tool to analyze LV diastolic function. Keren et al. (26) reported that diastolic dysfunction in patients with DCM was associated with disturbed left atrial filling due to both altered left atrial relaxation and reduced systolic motion of the mitral annulus. In keeping with these findings, we found a significantly reduced S/D ratio and increased A wave/pv-a ratio in patients with DCM as compared with normal subjects. In this study, severe MR impaired the systolic phase of pulmonary vein flow filling of the left atrium by elevating left atrial pressure, resulting in predominant atrial filling during diastole.

Color kinesis evaluation of global and regional diastolic function. Analysis of color kinesis images provides an additional tool for the assessment of global diastolic LV properties in patients with DCM. Analysis of LV area waveforms and their time derivatives obtained using acoustic quantification could provide essentially identical information on global LV filling, because color kinesis differs from acoustic quantification only in the manner by which endocardial motion is displayed. However, the major advantage of color kinesis over acoustic quantification is its ability to also assess regional endocardial motion. We developed an algorithm for analysis of color kinesis images, which in this study was applied to color kinesis images obtained in patients with DCM, to study both global and regional LV filling properties.

Among the color kinesis indexes of global LV diastolic properties, PFR was the most sensitive detector of global diastolic dysfunction in patients with DCM. A reduction in this index, which measures the velocity of endocardial expansion in early filling, may reflect a combined abnormal relaxation and altered compliance in patients with DCM. This finding was noted despite relatively increased early transmural peak filling velocity (E wave) secondary to MR. A reduced PFR was associated with a reduced mean filling time, in agreement with Ng et al. (36), who reported a shortened filling period as a mechanism of filling impairment in patients with DCM and MR. Thus, color kinesis indexes of global diastolic function appear to be less load-dependent than the conventional E/A ratio. Regression analysis showed that in our patients, color kinesis indexes also have a better predictive value for the diagnosis of diastolic dysfunction as compared with Doppler echocardiography.

We also studied regional LV diastolic properties using color kinesis by measuring the regional filling fraction and regional...
filling rate, and we found evidence of diastolic dysfunction in all segments. Brutsaert et al. (37) suggested that LV relaxation is a nonuniform process, and that this heterogeneity may be augmented by different disease states. For example, the peak early flow propagation velocity in the canine heart was found to reach the apex significantly later during induced regional myocardial ischemia (38). Studies with radionuclide angiography have demonstrated heterogeneous relaxation in normal subjects (39) and increased LV asynchrony in patients with hypertrophic cardiomyopathy (40) and coronary disease (41). However, the clinical significance of nonuniform diastolic endocardial motion has been difficult to determine owing to the lack of easy measurement techniques.

In this study, we used color kinesis images to assess regional heterogeneity in diastolic wall motion. To determine the feasibility of this approach, we initially studied patients with DCM and LBBB, because they are known to have diastolic dysfunction in the interventricular septum (42). The asynchrony index calculated from color kinesis images reflected an expected significant increase in diastolic wall motion asynchrony (Fig. 4, Table 2), thus demonstrating the feasibility of using this new technique to detect differences in the timing of regional diastolic endocardial motion. Interestingly, patients with DCM and normal conduction had no increased asynchrony, with the exception of those with severe MR (Table 2). This observation suggests that increased preload may augment LV diastolic asynchrony, particularly during the mid-filling phase. Our findings are in agreement with those of Hayashida et al. (12), who demonstrated in patients with DCM that myocardial relaxation is sensitive to changes in loading conditions and regional nonuniformity, and that load reduction may improve both relaxation and systolic performance of the LV.

Effects of dobutamine and nitroprusside on LV diastolic properties. Our results obtained with the lusitropic and vasodilator agents demonstrated the ability of color kinesis to detect the differences between the effects of both drugs in patients with DCM, differences that were not readily evident by Doppler evaluation. The lusitropic effect of dobutamine resulted in an increase in PFR with a shortening of the time of LV filling, as compared with nitroprusside. Carrol et al. (43) demonstrated that nitroprusside, at doses similar to those used in this study, had no consistent effect on LV pressure decay. There are significant data suggesting that nitroprusside could have altered LV filling by modifying pericardial restraint or ventricular interaction, or both (43–47). In contrast, dobutamine, at the doses used, had a more profound effect on ventricular pressure decay than did nitroprusside (43). This effect is in agreement with the improvement in peak filling rate associated with faster LV filling, reflected by the reduced mean filling time observed in this study. In addition, the presence of MR in these patients could have influenced the changes in LV filling properties obtained with both drugs.

Our results demonstrated that diastolic asynchrony in patients with DCM is more pronounced during early LV filling, in agreement with a previous study based on invasive methodology (12). Interestingly, the administration of nitroprusside and dobutamine was associated with a trend toward normalization of global diastolic function indexes, and, accordingly, regional asynchrony during early filling decreased with these drugs.

Study limitations. Color kinesis, as well as other echocardiographic techniques, has its limitations. The accuracy of the measurements is influenced by image quality. Because this technique is based on acoustic quantification, it requires careful gain adjustments to accurately track endocardial boundaries; it is therefore operator dependent and, to a certain degree, subjective. However, these adjustments were performed using strictly defined guidelines previously described in detail (16). In contrast, the analysis procedure was performed

| Table 2. Asynchrony Index Obtained From Color Kinesis Images at Different Percentages of Left Ventricular Filling Time in Normal Subjects and in Different Subgroups of Patients With Dilated Cardiomyopathy |
|-----------------|-----------------|-----------------|
|                  | 25% Filling Time | 50% Filling Time | 75% Filling Time |
| Normal subjects (n = 24) | 10.2 ± 3.1      | 8.3 ± 3.3      | 5.5 ± 2.2      |
| Patients with DCM and LBBB (n = 5) | 15.0 ± 2.5*     | 14.0 ± 3.6*     | 8.0 ± 3.7*     |
| Patients with DCM but no LBBB (n = 19) | 11.1 ± 5.8     | 8.6 ± 3.7     | 5.7 ± 3.4     |
| MMR (n = 11) | 10.9 ± 5.6      | 7.3 ± 2.2      | 4.8 ± 1.5      |
| SMR (n = 8) | 11.4 ± 6.1       | 10.4 ± 4.5†    | 6.9 ± 4.7     |
| *p < 0.05 compared with normal subjects. †p < 0.05 compared with moderate mitral regurgitation. Data are presented as mean value ± SD. DCM = dilated cardiomyopathy; LBBB = left bundle branch block; MMR = moderate mitral regurgitation; SMR = severe mitral regurgitation. |

| Table 3. Effects of Nitroprusside and Dobutamine Infusion on Doppler and Color Kinesis Indexes of Left Ventricular Diastolic Function in Patients With Dilated Cardiomyopathy |
|-----------------|-----------------|-----------------|
|                  | Baseline        | Nitroprusside   | Dobutamine     |
| E/A ratio       | 2.6 ± 1.5       | 1.6 ± 0.8       | 1.8 ± 1.0      |
| IVRT (ms)       | 95 ± 15         | 70 ± 12         | 69 ± 11        |
| S/D ratio       | 0.7 ± 0.3       | 0.8 ± 0.4       | 0.8 ± 0.4      |
| Filling fraction (%EDA) | 32 ± 10       | 38 ± 8          | 33 ± 4         |
| PFR/HR (EDA/beat) | 2.2 ± 0.8     | 2.2 ± 0.5       | 2.3 ± 0.5      |
| MFT/RR          | 236 ± 32        | 254 ± 23        | 226 ± 29*      |

*p < 0.05 compared with nitroprusside. Data are presented as mean value ± SD. Abbreviations as in Table 1.
Cardiac translation and rotation may affect the evaluation of endocardial motion by color kinesis, which does not provide a correction for this potential source of error. Accordingly, a question could be raised whether our findings of increased diastolic asynchrony in patients with DCM are an artifact secondary to cardiac translation and rotation. However, translation and rotation are directly related to the contractile activity of the heart, which is reduced in patients with DCM. Nevertheless, the endocardial motion asynchrony we observed in these patients was increased as compared with normal subjects, despite the decreased translation and rotation.

The version of color kinesis used in this study had only 19 different hues available for color-encoding of diastolic endocardial motion, and therefore did not allow for color-encoding of the entire LV filling period in patients with heart rates <55 beats/min. Nevertheless, the results of this study add new evidence to the importance of the regional evaluation in the complex pathophysiology of diastolic dysfunction in patients with DCM.

In this study, we did not compare color kinesis measurements of regional LV endocardial motion with another objective independent technique, which is important to establish the clinical value of this technique. We could have correlated our findings with measurements made by LV ventriculography combined with high fidelity pressure recordings (12). However, this method is invasive and therefore could not be performed in patients in whom this test is not clinically indicated. In addition, this method disregards regional variations in diastolic pressure within the LV. Alternatively, we could have used radionuclide angiography to assess regional LV function. However, this method is limited to a single view and its accuracy was shown to be affected by a high level of regional counting fluctuations (41). Therefore, to the best of our knowledge, there is no established noninvasive “reference standard” technique for the accurate assessment of the timing of regional diastolic endocardial motion. Therefore, further prospective studies are required to elucidate the origin, role and natural history of regional asynchrony in this disease, as well as its response to therapy.

Conclusions. In this study, quantitative analysis of color kinesis images allowed us to demonstrate significant differences between indexes of LV diastolic function obtained in patients with DCM and those in normal subjects. This technique appears to provide less load-dependent evaluation of global LV diastolic function than Doppler echocardiography and can differentiate between the effects of lusitropic and vasodilator therapy. In addition, assessment of regional diastolic endocardial motion asynchrony using color kinesis was shown to be feasible and may in the future become a clinically useful tool for the diagnosis of diastolic dysfunction.

References


