Differentiation of Ischemic From Nonischemic Cardiomyopathy During Dobutamine Stress by Left Ventricular Long-Axis Function

Addison Effect of Left Bundle-Branch Block

Alison M. Duncan, MRCP; Darrel P. Francis, MD; Derek G. Gibson, FRCP; Michael Y. Henein, MD, PhD

Background—Resting regional wall-motion abnormalities do not reliably distinguish ischemic from nonischemic cardiomyopathy. Dobutamine stress echocardiography with use of the wall-motion score index (WMSI) identifies coronary artery disease (CAD) in dilated cardiomyopathy (DCM), but the technique is subjective and further complicated by left bundle-branch block (LBBB). Long-axis motion is sensitive to ischemia and can be assessed quantitatively. We aimed to compare long-axis function with WMSI for detecting CAD in DCM with or without LBBB.

Methods and Results—Seventy-three patients with DCM, 48 with CAD (16 with LBBB), and 25 without CAD (10 with LBBB) were studied. Long-axis M-mode, pulsed-wave tissue Doppler echograms (lateral, septal, and posterior walls), and WMSI were assessed at rest and at peak dobutamine stress. Failure to increase systolic amplitude (total amplitude minus post-ejection shortening) by 2 mm or early diastolic velocity by 1.1 cm/s was the best discriminator for CAD (systolic amplitude, sensitivity 85%, specificity 86%; lengthening velocity, 71% and 94%, respectively; \( P<0.001 \)). Both had greater predictive accuracy than did WMSI (sensitivity 67%, specificity 76%; \( P=0.001 \)). The predictive accuracy of changes in septal long-axis function was similar to those of average long-axis function (systolic amplitude cutoff \( =1.5 \) mm, lengthening velocity cutoff \( =1.5 \) cm/s). However in LBBB, systolic amplitude proved to be the only significant discriminator for CAD, with sensitivity and specificity reaching 94% and 100%, respectively (\( P<0.01 \) versus early diastolic lengthening velocity).

Conclusions—Quantified stress long-axis function identifies CAD in DCM with greater sensitivity and specificity than does standard WMSI, particularly in the presence of LBBB. (Circulation. 2003;108:1214-1220.)

Key Words: coronary disease ■ cardiomyopathy ■ bundle-branch block ■ echocardiography ■ imaging

Differentiating ischemic from nonischemic cardiomyopathy is important prognostically and therapeutically1 but might be difficult clinically. Regional wall motion at rest might be abnormal in as many as two thirds of patients with nonischemic cardiomyopathy, whereas patients with ischemic cardiomyopathy might have uniform hypokinesis.2 Dobutamine stress echocardiography and thallium scintigraphy detect coronary artery disease (CAD) in dilated cardiomyopathy with greater accuracy than does resting echocardiography,3–6 but both techniques might give conflicting results.7 Moreover, their performance in cases with coexisting left bundle-branch block (LBBB) has been disappointing; scintigraphy might give false-positive anteroseptal and septal perfusion defects,8 whereas conventional wall-motion analysis might be degraded.9 Inclusion of patients with LBBB in the assessment of ischemia is thus important if broad clinical validity is sought.

Longitudinal myocardial fibers are predominately subendocardial and are sensitive to ischemia9 and abnormal activation.10 Furthermore, stress-induced changes in long-axis function can be measured objectively.11,12 Our study thus aimed to compare long-axis behavior at rest and during stress in ischemic and nonischemic cardiomyopathy, with and without LBBB, and thus define criteria for differentiating between them.

Methods

Study Population

We studied 73 patients from a heart failure clinic (mean±SD age, 63±10 years; 63 men). All had a left ventricular (LV) end-diastolic dimension >5.6 cm and an end-systolic dimension >4.0 cm on M-mode echocardiograms but were otherwise unselected. Coronary angiography was performed within 3 months of stress echocardiography. Significant CAD was defined as >70% reduction in the absolute lumen diameter of a major epicardial artery or major branch...
vessel. LBBB was diagnosed on the basis of a QRS duration >120 ms; absent q waves; wide, slurred R waves in V1 and V6; and monophasic QS or rS waves in V1 and V6. Forty-eight patients had ischemic cardiomyopathy (16 with LBBB), and 22 had a normal coronary arteriogram (nonischemic cardiomyopathy; 10 with LBBB). Patients with recent-onset heart failure (>14 days), myocardial infarction (<3 months), unstable angina (<1 month), structural valve disease, ventricular tachycardia, or atrial fibrillation were excluded. At enrolment, 41 patients with ischemic cardiomyopathy were receiving an angiotensin-converting enzyme inhibitor, 38 a diuretic, and 18 a β-blocker; 24 patients with nonischemic cardiomyopathy were receiving an angiotensin-converting enzyme inhibitor, 24 a diuretic, and 7 a β-blocker. Patients were compared with 20 control subjects of similar age (mean±SD, 58±11 years; 8 men), none with a history of angina, hypertension, or diabetes. The Royal Brompton and Harefield Ethics Committee approved the study protocol. All subjects gave written, informed consent, and there were no complications related to the investigation.

**Dobutamine Stress Echocardiography**

Dobutamine stress echocardiography was performed with concurrent 12-lead ECG, phonocardiogram, and blood pressure monitoring. Transesophageal echocardiography was performed with an echocardiographic system (Hewlett-Packard Sonos 5500), a multifrequency transducer, and harmonic imaging as appropriate. Two-dimensional echocardiography was performed from the parasternal long- and short-axis views and apical 4- and 2-chamber views. LV minor-axis dimensions were measured at end diastole (onset of the QRS complex) and at end systole (A2 on the phonocardiogram) from a parasternal, 2-dimensional, guided M-mode echocardiogram, with the cursor at the tips of the mitral valve leaflets. Mitral regurgitation (MR) was graded by standard criteria (mild, moderate, or severe) according to the distance from the valve orifice that the regurgitant jet remained detectable on the color flow Doppler recording.

**Measurements**

LV long-axis, pulsed-wave, tissue Doppler (PWTD) and M-mode recordings were obtained with the cursor positioned at the lateral, septal, and posterior angles of the mitral ring. A Doppler velocity range of ±30 cm/s was selected to display systolic and early diastolic velocities. Total amplitude was defined as maximum displacement of the ring between the onset of QRS and peak inward movement at or after A2 (identified from the valve-closure artifact on aortic Doppler). Postejection shortening (PES) was measured as the amplitude of shortening after A2. Systolic amplitude, representing long-axis displacement during ventricular ejection, was calculated by subtracting PES from total amplitude. All PWTD and M-mode recordings were obtained at a paper speed of 100 mm/s. To make data comparable with previous studies of wall-motion score index (WMSI), measurements from the 3 long-axis sites were combined for PWTD and M-mode. Because LBBB is frequently associated with asynchronous septal contraction, septal long-axis values are also presented separately.

**Wall Motion Score Index**

According to the American Society of Echocardiography criteria, the LV was divided into 16 segments, and WMSI (summation of all segments scored divided by the number of segments scored) was calculated. Segmental wall motion was graded 1 to 4 (where 1=normal wall motion at rest and hyperkinesis with stress, 2=hy-pokinesis at rest or segments with normal amplitude at rest but a reduction with dobutamine, 3=akinesis, and 4=dyskinesis).

**Statistical Analysis**

Differences between groups are reported as mean±SD. Rest and stress values within each study group were compared with a paired Student’s t test. An unpaired Student’s t test was used to compare values between groups. In view of multiple t tests, a significant difference was set at P<0.01. The incremental value of measurements of long-axis function and WMSI were tested with a stepwise logistic regression, and a receiver operating characteristic curve was constructed to establish the sensitivity and specificity of a range of threshold changes in long-axis and WMSI values for angiographic CAD.

**Reproducibility**

An independent investigator, unaware of the clinical history or angiographic data, analyzed all echocardiographic images. Duplicate determinations of long-axis measurements and WMSI were assessed in 20 patients. Within- and between-observer values were determined independently by a third individual, who was also blinded to the original diagnosis. Reproducibility was expressed as the root mean square (RMS) difference between duplicate values. The interobserver RMS difference was 0.6 mm for systolic amplitude, 0.3 cm/s for systolic velocity, 0.2 cm/s for early diastolic velocity, and 0.22 for WMSI. The interobserver RMS difference was 0.7 mm, 0.4 cm/s, 0.2 cm/s, and 0.24, respectively. The RMS of the difference between observers for stress-induced change in systolic amplitude was 0.5 mm, and the corresponding figure for WMSI was 0.15.

**Results**

**LV Dysfunction**

LV dysfunction was equally severe in nonischemic and ischemic cardiomyopathy when activation was normal (Table 1). Patients with both CAD and LBBB had significantly greater cavity dilatation and resting WMSI (Table 2) than did patients with either alone (all P<0.01). The extent of CAD was not different in subgroups of ischemic cardiomyopathy. Peak dobutamine dose and peak heart rate were similar between subgroups, but all patients with ischemic cardiomyopathy developed symptoms or ECG changes during stress, unlike the majority with nonischemic cardiomyopathy (Table 1).

**Changes in Average Long-Axis Function With Stress**

In nonischemic cardiomyopathy (Table 2), the average long-axis response to stress was similar to that of control subjects, irrespective of LBBB (Figure 1). Total amplitude, systolic amplitude, systolic velocity, and early diastolic velocity all increased (all P<0.01); PES did not appear in any patient; and in those with LBBB, PES amplitude fell (P<0.01). In ischemic cardiomyopathy (Table 2), systolic amplitude and early diastolic velocity failed to increase, and PES was exaggerated (P<0.001). Systolic velocity did increase, but by less than in nonischemic cardiomyopathy (P<0.01). With LBBB, systolic amplitude, systolic velocity, and early diastolic velocity increased even less with stress, whereas PES was greater (all P<0.01).

**Changes in Septal Long-Axis Function With Stress**

In nonischemic cardiomyopathy and LBBB (Table 3), the septal long-axis response to stress was similar to that in control subjects. By contrast, septal systolic amplitude and velocities failed to change in ischemic cardiomyopathy and LBBB (Table 3), yet PES amplitude, and hence total amplitude, increased (P<0.01).

**Global WMSI**

Global WMSI fell with stress in nonischemic cardiomyopathy and normal activation (P<0.001) but did not change in any other subgroup (Table 2).
TABLE 1. Clinical Details of Study Population

<table>
<thead>
<tr>
<th>Variable</th>
<th>Nonischemic Cardiomyopathy</th>
<th>Ischemic Cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Controls (n=20)</td>
<td>NA (n=15)</td>
</tr>
<tr>
<td></td>
<td>NA (n=32)</td>
<td>LBBB (n=16)</td>
</tr>
<tr>
<td>EDD, mm</td>
<td>50±4</td>
<td>66±7</td>
</tr>
<tr>
<td>ESD, mm</td>
<td>33±6</td>
<td>54±5</td>
</tr>
<tr>
<td>Three-vessel CAD, n</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Two-vessel CAD, n</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Stress end point</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dobutamine dose, µg/kg per minute</td>
<td>39±2</td>
<td>37±4</td>
</tr>
<tr>
<td>Peak heart rate, bpm</td>
<td>117±12</td>
<td>118±20</td>
</tr>
<tr>
<td>Chest pain, n</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Breathlessness, n</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Ventricular ectopic beats, n</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Hypertension, n</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>ST-segment depression, n</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>T-wave inversion, n</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Values are mean±SD. NA indicates normal activation; EDD, end-diastolic dimension; and ESD, end-systolic dimension.

*P<0.001, ischemic cardiomyopathy (LBBB) vs nonischemic cardiomyopathy (LBBB).
†P<0.01, ischemic cardiomyopathy: LBBB vs NA.

Functional MR
MR was detected at rest in all patients, but its severity did not worsen in any patient during stress. Moderate MR became mild in 27 (18 ischemic), mild MR regressed completely in 35 (24 ischemic), and MR severity did not change in 11 (6 ischemic).

TABLE 2. Average Long-Axis Function

<table>
<thead>
<tr>
<th>Variable</th>
<th>Nonischemic Cardiomyopathy</th>
<th>Ischemic Cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Controls (n=20)</td>
<td>NA (n=15)</td>
</tr>
<tr>
<td></td>
<td>NA (n=32)</td>
<td>LBBB (n=16)</td>
</tr>
<tr>
<td>Rest</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TA, mm</td>
<td>13.8±1.7</td>
<td>11.9±1.9*</td>
</tr>
<tr>
<td>SA, mm</td>
<td>13.8±1.7</td>
<td>11.9±1.9*</td>
</tr>
<tr>
<td>PES, mm</td>
<td>0±0</td>
<td>0±0</td>
</tr>
<tr>
<td>SV, cm/s</td>
<td>7.8±1.5</td>
<td>4.8±1.4**</td>
</tr>
<tr>
<td>EDV, cm/s</td>
<td>7.3±1.9</td>
<td>5.6±1.3*</td>
</tr>
<tr>
<td>WMSI</td>
<td>...</td>
<td>2.48±0.22</td>
</tr>
<tr>
<td>Response to stress</td>
<td></td>
<td></td>
</tr>
<tr>
<td>∆ TA, mm</td>
<td>+4.0±0.7††</td>
<td>+3.1±1.5†</td>
</tr>
<tr>
<td>∆ SA, mm</td>
<td>+4.0±0.7††</td>
<td>+3.1±1.5†</td>
</tr>
<tr>
<td>∆ PES, mm</td>
<td>0±0</td>
<td>0±0</td>
</tr>
<tr>
<td>∆ SV, cm/s</td>
<td>+4.3±1.6††</td>
<td>+3.8±2.3††</td>
</tr>
<tr>
<td>∆ EDV, cm/s</td>
<td>+2.4±0.9††</td>
<td>+4.8±2.7††</td>
</tr>
<tr>
<td>∆ WMSI</td>
<td>...</td>
<td>−0.40±0.33§</td>
</tr>
</tbody>
</table>

Values are mean±SD. NA indicates normal activation; TA, total amplitude; SA, systolic amplitude; SV, systolic velocity; and EDV, early diastolic velocity.

*P<0.01. **P<0.001, nonischemic cardiomyopathy (NA) vs controls at rest.
†P<0.01. ††P<0.001, nonischemic cardiomyopathy: LBBB vs NA at rest.
‡P<0.01. ‡‡P<0.001, ischemic cardiomyopathy (NA) vs nonischemic cardiomyopathy (NA) at rest.
§P<0.01. §§P<0.001, ischemic cardiomyopathy (LBBB) vs ischemic cardiomyopathy (NA) at rest.
¶P<0.01. †¶P<0.001, stress vs rest within group.

Prediction of CAD From Changes in Average Long-Axis and WMSI Values With Stress
Stress-induced changes in average long-axis amplitude, shortening, and early diastolic lengthening velocities and in WMSI were all univariate predictors of CAD (Table 4). The best discriminators, however, were changes in systolic amplitude...
and early diastolic lengthening velocity (both \( P < 0.001 \)). An increment of 2 mm in systolic amplitude correctly identified 41 of 48 patients with CAD and 22 of 25 patients without CAD (sensitivity 85%, specificity 88%; Figure 2), whereas an increment of 1.1 cm/s in early diastolic lengthening velocity identified 34 of 48 patients with CAD and 23 of 25 patients without (sensitivity 71%, specificity 94%). The predictive accuracy of both systolic amplitude and early diastolic velocity for detecting CAD was significantly greater than that of changes in WMSI (\( P < 0.001 \); Figure 3). In the LBBB group, the same cutoff criteria gave a sensitivity and specificity of 88% and 89% (systolic amplitude), 70% and 88% (early diastolic lengthening velocity; difference = NS), and 67% and 76% for WMSI (\( P < 0.001 \) versus systolic amplitude), respectively.

**Prediction of CAD From Septal Long-Axis Values**
Changes in septal systolic amplitude in the study population had a predictive accuracy similar to that of average long-axis function, with systolic amplitude (cutoff = 1.5 mm) and early diastolic lengthening velocity (cutoff = 1.5 cm/s) being the best discriminators (Table 4 and Figure 3). However, in the LBBB group, systolic amplitude proved to be the only significant discriminator for CAD (cutoff = 1.5 mm), with sensitivity and specificity reaching 94% and 100%, respectively (\( P < 0.01 \) versus early diastolic lengthening velocity; Figure 3).

**Discussion**

**Significance of Stress-Induced Changes**
Stress-induced changes in total long-axis amplitude, systolic long-axis velocities, and WMSI all distinguished ischemic...
Failure to increase systolic amplitude by 2 mm or early systolic amplitude and early diastolic lengthening velocity. The best overall predictors of CAD, however, were changes in long-axis variables from nonischemic cardiomyopathy. The best overall predictors of CAD, however, were changes in long-axis systolic amplitude and early diastolic lengthening velocity. Failure to increase systolic amplitude by 2 mm or early diastolic lengthening velocity by 1.1 cm/s with stress, even when LBBB was present, had a greater predictive accuracy than did changes in WMSI. In LBBB, failure to increase septal systolic amplitude by >1.5 mm with stress was the only discriminator for CAD and was highly sensitive and specific.

### Table 3. Septal Long-Axis Function

<table>
<thead>
<tr>
<th>Variable</th>
<th>Controls (n=20)</th>
<th>Nonischemic Cardiomyopathy</th>
<th>Ischemic Cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>NA (n=15)</td>
<td>LBBB (n=10)</td>
</tr>
<tr>
<td>TA, mm</td>
<td>13.2±2.0</td>
<td>11.5±2.8</td>
<td>6.4±1.6†</td>
</tr>
<tr>
<td>SA, mm</td>
<td>13.2±2.0</td>
<td>11.5±2.8</td>
<td>4.6±1.8†</td>
</tr>
<tr>
<td>PES, mm</td>
<td>0±0</td>
<td>0±0</td>
<td>1.8±0.9†</td>
</tr>
<tr>
<td>SV, cm/s</td>
<td>6.9±1.6</td>
<td>3.5±1.0*</td>
<td>3.3±0.9</td>
</tr>
<tr>
<td>EDV, cm/s</td>
<td>5.7±1.4</td>
<td>5.2±1.8</td>
<td>4.6±2.2</td>
</tr>
</tbody>
</table>

Response to stress

- Δ TA, mm: +3.5±2.4‡, +3.0±1.4†, +1.3±0.5||, +1.0±1.7‡, +1.3±1.6||
- Δ SA, mm: +3.5±2.4‡, +3.0±1.4†, +2.7±1.0||, -0.3±1.9‡, +0.4±0.2||
- Δ PES, mm: 0±0, 0±0, -1.4±1.0||, +1.5±1.3†, +0.9±1.2‡
- Δ SV, cm/s: +4.4±1.8†, +3.8±1.7†, +3.1±2.4‡, +1.8±2.0*, +1.4±1.7
- Δ EDV, cm/s: +2.8±1.7†, +3.9±1.8†, +2.4±1.7, +1.3±1.5‡, +0.6±2.7

Values are mean±SD. NA indicates normal activation; TA, total amplitude; SA, systolic amplitude; SV, systolic velocity; and EDV, early diastolic velocity.

- P<0.001, nonischemic cardiomyopathy (NA) vs controls at rest.
- †P<0.01, ischemic cardiomyopathy (NA) vs nonischemic cardiomyopathy (NA) at rest.
- §P<0.001, ischemic cardiomyopathy (LBBB) vs ischemic cardiomyopathy (NA) at rest.
- ¶P<0.01, ¶¶P<0.001, stress vs rest within group.

### Table 4. Predictors of CAD

<table>
<thead>
<tr>
<th>Variable</th>
<th>AUC (CI)</th>
<th>Cutoff</th>
<th>χ²</th>
<th>P</th>
<th>Sensitivity, %</th>
<th>Specificity, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δ Average SA, mm</td>
<td>+4.1</td>
<td>0.92 (0.83–0.97)</td>
<td>+2.0</td>
<td>16.9</td>
<td>&lt;0.001</td>
<td>85</td>
</tr>
<tr>
<td>Δ Average SV, cm/s</td>
<td>+3.4</td>
<td>0.79 (0.65–0.90)*</td>
<td>+2.0</td>
<td>11.4</td>
<td>&lt;0.001</td>
<td>92</td>
</tr>
<tr>
<td>Δ Average EDV, cm/s</td>
<td>+3.2</td>
<td>0.87 (0.73–0.95)</td>
<td>+1.9</td>
<td>10.4</td>
<td>&lt;0.001</td>
<td>71</td>
</tr>
<tr>
<td>Δ Average TA, mm</td>
<td>+3.0</td>
<td>0.72 (0.60–0.82)**</td>
<td>+2.0</td>
<td>8.8</td>
<td>0.003</td>
<td>75</td>
</tr>
<tr>
<td>Δ WMSI</td>
<td>-2.7</td>
<td>0.69 (0.58–0.80)**</td>
<td>-0.11</td>
<td>7.5</td>
<td>0.006</td>
<td>67</td>
</tr>
</tbody>
</table>

**Septal predictors of CAD**

- All patients (n=73)
  - Δ Septal SA, mm | +3.9 | 0.92 (0.83–0.97) | +1.5 | 15.1 | <0.001 | 80 | 100 |
  - Δ Septal SV, cm/s | +2.8 | 0.72 (0.57–0.85)† | +1.8 | 9.9 | <0.01 | 75 | 76 |
  - Δ Septal EDV, cm/s | +2.9 | 0.82 (0.68–0.92) | +1.5 | 8.0 | <0.01 | 63 | 91 |
  - Δ Septal TA, mm | +3.1 | 0.74 (0.62–0.83)† | +0 | 9.6 | <0.01 | 40 | 100 |
- LBBB (n=26)
  - Δ Septal SA, mm | +2.5 | 0.97 (0.83–0.98) | +1.5 | 6.2 | <0.01 | 94 | 100 |
  - Δ Septal SV, cm/s | +1.6 | 0.66 (0.40–0.87)† | +1.8 | 1.8 | NS | 78 | 67 |
  - Δ Septal EDV, cm/s | +1.5 | 0.69 (0.48–0.85)† | +1.5 | 2.2 | NS | 67 | 78 |
  - Δ Septal TA, mm | +0.7 | 0.64 (0.43–0.82)† | +0 | 0.5 | NS | 38 | 100 |

* indicates coefficient/SE; AUC, area under curve; CI, 95% confidence interval; SA, systolic amplitude; SV, systolic velocity; EDV, early diastolic velocity; and TA, total amplitude.

*P<0.01, **P<0.001, vs AUC for average SA.
†P<0.01, vs AUC for septal SA (all patients).
‡P<0.01, vs AUC for septal SA (LBBB alone).
Mechanisms
The greater sensitivity of changes in long-axis function over WMSI might have several explanations. The mitral ring gives rise to a strong echo, even when image quality is suboptimal, so that systolic amplitude and PWTD signals are simple to quantify even in patients with LBBB, thereby providing objective and reproducible measurements. In contrast, WMS analysis remains semiquantitative and dependent on operator experience. A further advantage of long-axis assessment is that incoordinate long-axis shortening after aortic valve closure can be distinguished from that during ejection, an assessment that is not possible with the repetition rate of 2-dimensional imaging. The sensitivity and specificity of changes in septal systolic amplitude and early diastolic lengthening velocity were similar when all patients were considered, findings that are compatible with a previously demonstrated correlation between them. Finally, the contrasting effects of stress in LBBB with or without ischemia meant that septal long-axis measurements performed particularly well in this group. This probably reflected their direct relation to QRS shortening with stress in nonischemic but not in ischemic cardiomyopathy. In this group, PWTD proved less satisfactory, the low amplitude and velocities probably favoring M-mode.

Limitations
Our results represent a learning set, but our cutoff values for long-axis amplitude and WMSI are similar to those previously reported. Our patients had either multivessel CAD or none at all, reflecting the low incidence of patients with heart failure and single-vessel coronary disease. As in the literature, patients with significant ventricular arrhythmias and atrial fibrillation were not included. The sensitivity of WMSI in previous studies for detecting CAD in dilated cardiomyopathy ranged between 26% and 100%, and our levels were similar. However, our figures for detecting CAD in LBBB by using WMSI were somewhat lower than previously reported, probably because of the associated hypokinesis. Strain-rate imaging might constitute another useful method for analyzing regional LV function in this setting.

Figure 2. Changes in average long-axis systolic amplitude (top), average early diastolic lengthening velocity (EDV, middle), and WMSI (bottom) between rest and stress in dilated cardiomyopathy (DCM). Failure to increase average systolic amplitude by 2 mm or EDV by 1.5 cm/s was more sensitive and more specific than WMSI.
but this technique is not yet widely available, and its predictive accuracy in patients with LBBB has not been studied. Functional MR was detected at rest in all patients, but as previously reported, its severity did not worsen with stress. Finally, to distinguish septal systolic amplitude from early diastolic lengthening velocity, the performance of stress. Finally, to distinguish septal systolic amplitude from early diastolic lengthening velocity.

Conclusions

Long-axis dobutamine stress M-mode and PWTD echocardiographic methods are quantifiable, reproducible, and non-invasive techniques for assessing the effects of stress in dilated cardiomyopathy. They differentiate ischemic from nonischemic cardiomyopathy with greater sensitivity and specificity than does the WMSI and thus, might be a useful and simple adjunct to standard dobutamine stress testing for detecting CAD in patients with dilated cardiomyopathy, particularly when LBBB is present.

Acknowledgment

Dr A. Duncan was supported by the Garfield Weston Trust.

References

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