Effects of physiologic maneuvers on cardiac performance and mitral regurgitation in patients with dilated cardiomyopathy

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Objective We observed systolic function by using Doppler echocardiography during valsalva (VM), handgrip, standing and leg elevation maneuvers in patients with mitral regurgitation (MR).

Methods Patient population consisted with 32 patients with dilated cardiomyopathy. Patient population was consisted of only men and average age are 60 years. They were in functional class II to III according to the New York Heart Association classification. None of the patients had primary mitral valve disease, flail mitral leaflet or aortic regurgitation. Left ventricular ejection fraction averaged 38±12 %. Subjects with technically unsatisfactory echocardiograms were excluded from the study. Left ventricular end diastolic volume was substantially over in all patients.

Introduction
In clinical practice, we can use commonly physiologic maneuvers to identify cardiac murmur in patient with heart disease. Some of maneuvers were also previously use to diagnose hypertrophic cardiomyopathy (1-5). Doppler echocardiography as a noninvasive procedure is superior for detection cardiac response to physiologic maneuvers.

The current study tests that physiologic maneuvers to determine systolic function are more precisely by creating changes in hemodynamic status in patient with heart disease.

Material and Method
This study group was composed of 32 patients who had mitral regurgitation (MR). All of them had dilated cardiomyopathy (DCMP) and sinus rhythm except one who was in atrial fibrillation. Patient population was consisted of only men and average age 60, range 30 to 85 years. Twenty-seven of the 32 patients had an apical holosystolic murmur when examined by cardiologist. The murmur was detected easily with phonocardiographic examination. They were in functional class II to III according to the New York Heart Association classification. None of the patients had primary mitral valve disease, flail mitral leaflet or aortic regurgitation. Left ventricular ejection fraction (LVEF) averaged 38±12 %. Subjects with technically unsatisfactory echocardiograms were excluded from the study. Left ventricular end diastolic volume (LVEDV) was substantially over in all patients. In twenty two patients, the etiology of the DCMP was coronary artery disease that was documented by previous myocardial infarctions and abnormal coronary angiograms.

Maneuvers: Control measurements were recorded before 4 sequential maneuvers; valsalva maneuvers (VM), leg cleration (LE), handgrip (HG) and standing. VM was performed by constant exhalation against 40 mmHg pressure which facilitated to obtain volumes, approximately equal to the end expiratory lung volume. The maneuvers were generally repeated two times to complete the ultrasonic measurement in which the recordings were obtained during the strain phase 2. For LE maneuver, we took echocardiographic measurements while the patient’s legs flexed in the position of the lower legs horizontal to the table. Left HG was performed with a dynamometer at 40% of maximal grip to evaluate the changes of patients’ echocardiographic parameters during the handgrip maneuvers. Finally values during standing maneuvers were obtained while patients were standing with their left elbows flexed. This position was maintained by using an elevated stand to keep left elbows flexed. The aim of the flexion was to displace the left ventricular apex anteriorly. Doppler echocardiography: Acuson-128

Table I. Systolic parameters by using doppler echocardiography in patients with dilated cardiomyopathy.

<table>
<thead>
<tr>
<th>Variables</th>
<th>C</th>
<th>VM</th>
<th>LE</th>
<th>HG</th>
<th>ST</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR(b/m)</td>
<td>74±22</td>
<td>81±15</td>
<td>72±16</td>
<td>82±21</td>
<td>78±16</td>
</tr>
</tbody>
</table>

Accepted for publication: 13 April 1996

1998 Eastern Journal of Medicine
BP(mmHg) 122±18 105±20 123±16 148±17 120±16
LVEDV(ml) 181±38 145±42 184±43 186±42 150±39
LVESV(ml) 26±18 20±11 28±13 43±20 20±17
TSV(ml) 70±22 50±25 72±23 79±21 54±18
ASV(ml) 43±19 20±18 44±21 36±17 33±18
MRV(ml) 26±18 20±11 28±13 43±20 20±17
RF(%) 38±15 42±14 38±15 54±18 37±15
MRA(cm²) 6.6±0,9 4.3±0,7 6.7±0,9 11.9±1 4.4±0,8
EF 38±12 35±13 35±12 41±15 35±13

Table II. Percentage changes from mean during maneuvers in patients with dilated cardiomyopathy.

<table>
<thead>
<tr>
<th>Variables</th>
<th>VM</th>
<th>LE</th>
<th>HG</th>
<th>ST</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>+5</td>
<td>-1</td>
<td>+5</td>
<td>+3</td>
</tr>
<tr>
<td>BP</td>
<td>+6</td>
<td>+1</td>
<td>+10*</td>
<td>-1</td>
</tr>
<tr>
<td>LVEDV</td>
<td>-11*</td>
<td>+1</td>
<td>+2</td>
<td>-9*</td>
</tr>
<tr>
<td>LVESV</td>
<td>-6</td>
<td>+1</td>
<td>+2</td>
<td>-6</td>
</tr>
<tr>
<td>TSV</td>
<td>-16*</td>
<td>+1</td>
<td>+7*</td>
<td>-14*</td>
</tr>
<tr>
<td>ASV</td>
<td>-36*</td>
<td>+1</td>
<td>-9*</td>
<td>-13*</td>
</tr>
<tr>
<td>MRV</td>
<td>-15*</td>
<td>+1</td>
<td>+23*</td>
<td>-15*</td>
</tr>
<tr>
<td>RF</td>
<td>+5</td>
<td>+1</td>
<td>+17*</td>
<td>-1</td>
</tr>
<tr>
<td>MRA</td>
<td>-21*</td>
<td>+1</td>
<td>+28*</td>
<td>-20*</td>
</tr>
<tr>
<td>EF</td>
<td>-4</td>
<td>-4</td>
<td>+4</td>
<td>-4</td>
</tr>
</tbody>
</table>

*: p<0.05


The endocardial echoes were traced with the integrated Echo-Doppler analyzer (Freeland Cineloop system). TSV and ejection fraction (EF) was calculated from end-diastolic and end-systolic volumes. Forward stroke volume (ASV) by pulsed wave Doppler echocardiography: Forward aortic flow volume was calculated as the product of the integral of aortic outflow velocity and the cross sectional area of the aortic anulus. Regurgitation fraction (RF) is that fraction or percentage of TSV in incompetent mitral valve.

Statistics: The results are expressed as mean±standard deviation, and were consider significant at p<0.05. Percentage change is defined as maneuver -control/ mean. Hemodynamic and Doppler echocardiographic data at control and during maneuvers were tested for significance using Student's paired -t test. Regression analysis was performed according to standard procedures.

Results

Table I showes systolic and diastolic parameters by mean and ± standard deviation during maneuvers. Percentage changes from mean during maneuvers is presented table II. Heart rate did not change during these maneuvers. Blood pressure increased significantly with HG because of increased afterload. LVEDV decreased significantly by associated with decreasing preload during VM and standing position. TSV riased significantly with HG while it decreased with VM and standing maneuvers. LE did not significantly increase TSV. During phase 2 of VM , a 16 % drop in TSV associated with a 11% decrease in LVEDV. MRA diminished 21% during the VM . During HG, effort produced 10% increase in systolic blood pressure as well as a 7% increase in TSV and 23% in MRV. An important point is that there is significant rise in TSV during HG without significant changing with LVEDV and left ventricular endsystolic volume. Aortic stroke volume derived from aortic velocity time integral signal, declined significantly during VM, HG and standing position. MRV defined as stroke volume - aortic stroke volume increased significantly with HG. RF derived from MRV/ TSV ratio increased significantly by only HG. MRA changed significantly in all maneuvers except LE and showed good correlation with MRV, as showed
Figure-1. The response of standing maneuver decreased TSV 14% associated with a 9% reduction in LVEDV and 15% drop in MRV. The 1% increase in TSV and MRV due to LE was not significant. LVEF didn’t change during maneuvers in patient with MR.

Discussion
The normal response to bedside maneuvers can alter with loading conditions (5). In previous studies it was shown that VM and HG exercises could change the hemodynamic response in patient population (1,2,3,4). In present study it was investigated that these maneuvers in patients with DCMP changed normal hemodynamics response and mitral regurgitation. As it was presented in table-I, during the VM, heart rate and blood pressure did not change significantly when compared with control. LVEDV and MRV decreased significantly (-11%, -15% respectively) due to decreased venous return. Both TSV and ASV decreased significantly (-16% and -36%, respectively). Little et al (2), observed that stroke volume and LVEF did not change during the VM in patients with cardiomyopathy. Their patients had congestive heart failure with 23% EF. Average LVEF of patients was 38 % in our study. They performed VM lasting 15 seconds. We performed VM for 30 seconds. Our hemodynamic measurements started just within 20 seconds during VM. Perhaps the differences could be originated from patients clinical condition and duration of VM. So, MRV decreased significantly (-15 %) because of abated venous return. Effect of isometric exercise on cardiac performance and MR both in patients and normal subjects was investigated in previous study (3,7). Keren et al. found that LVEDV and LVESV did not change thought ASV measured by Doppler echocardiography decreased significantly (3) during HG. Our present data confirm the findings of Keren et al. We observed that the rise in systemic arterial pressure induced by isometric exercise may be caused by increase systemic peripheric vascular resistance than ASV decreased. Despite unchanging both LVEDV and LVESV, TSV increased significantly (+7 %). The augmented MR and costant of ventricular contractility explains these results. The rapid rise in systemic arterial pressure, is likely to worsen or even create functional MR. Boltwood et al (8). suggested that MR induced by HG did not directly correlated with ventricular dimension. MR can be associated with regurgitant orifice area which is related to left atrial volume.

Keren et al (3) explained that contractility of the left ventricle had characterized by the relation between stroke work and filling pressure. Calculation of stroke work should include the MRV which is parallel to fall in ASV in these patients. MR is a dynamic process and worsens when cardiac afterload is increased (6).

LE failed to alter hemodynamic status and systolic function. Standing affected systolic parameters like VM, because of decreased preload.

In conclusion, our findings demonstrate that bedside maneuvers affected the systolic function and severity of MR due to loading conditions.

References

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