PATHOPHYSIOLOGY AND NATURAL HISTORY
CARDIOMYOPATHY

Altered effect of the Valsalva maneuver on left ventricular volume in patients with cardiomyopathy

WILLIAM C. LITTLE, M.D., W. KENT BARR, M.D., AND MICHAEL H. CRAWFORD, M.D.

With the technical assistance of K. Wray Amon, B.S.

ABSTRACT The failure of the blood pressure to fall during the late strain phase of the Valsalva maneuver in patients with heart failure may result from the left ventricle operating on a flat portion of its function curve or from maintenance of left ventricular volume despite decreased systemic venous return. To test these possibilities, we studied the effect of the Valsalva maneuver (40 cm H2O for 15 sec) on left ventricular volume in 12 normal subjects with a mean left ventricular ejection fraction of 0.65 ± 0.07 (±SD) and in eight patients with nonischemic cardiomyopathy, evidence of pulmonary congestion, and a mean left ventricular ejection fraction of 0.23 ± 0.09. Left ventricular volume and right ventricular area were determined by apical two-dimensional echocardiography. In both groups the right ventricular end-diastolic area decreased during the late strain phase of the Valsalva maneuver. In normal subjects it decreased from 9.3 ± 1.5 to 5.6 ± 1.6 cm² (p < .001) and in patients it decreased from 13 ± 2.2 to 10 ± 2.9 cm² (p < .001). In normal subjects, left ventricular end-diastolic volume decreased from the control level during the Valsalva maneuver, and this was apparent in both the four-chamber (96 ± 21 to 68 ± 18 ml, p < .01) and two-chamber views (97 ± 15 to 56 ± 20 ml, p < .01). In the patients, left ventricular end-diastolic volume was not significantly different from control in either view (199 ± 70 to 195 ± 78 and 214 ± 77 to 218 ± 86 ml, respectively). In normal subjects, a decrease in stroke volume from control during the Valsalva maneuver was evident in both views (61 ± 13 to 40 ± 11 ml, p < .01 and 63 ± 9 to 33 ± 16 ml, p < .01), but in the patients there was no change in stroke volume in either view during the Valsalva maneuver (45 ± 21 to 45 ± 23 and 49 ± 12 to 49 ± 17 ml). We conclude that in patients with pulmonary congestion and reduced left ventricular ejection fraction, left ventricular stroke volume does not fall during the strain phase of the Valsalva maneuver because left ventricular end-diastolic volume is maintained.


AN ALTERED RESPONSE to the strain of the Valsalva maneuver has been noted in patients with congestive heart failure and the maneuver has been suggested as a bedside technique to detect left ventricular dysfunction. Patients with congestive heart failure, in contrast to normal subjects, maintain mean arterial pressure and pulse pressure throughout the strain phase of the Valsalva maneuver, indicating a preserved left ventricular stroke volume. Two mechanisms have been suggested to explain this abnormal response in patients with congestive heart failure: (1) The depressed left ventricle may operate on the flat portion of its function curve, in which case, stroke volume would remain unchanged despite a decrease in left ventricular end-diastolic volume during the strain of the Valsalva maneuver. This effect may be augmented by a decreased effective afterload resulting from the increase in intrathoracic pressure. (2) The left ventricular end-diastolic volume may be maintained in such patients during the strain and thus the stroke volume is unchanged.

To test these possibilities we studied the effect of a standardized Valsalva maneuver on right ventricular end-diastolic cross-sectional area, left ventricular end-diastolic volume, and left ventricular stroke volume determined by two-dimensional echocardiography in normal volunteers and patients with nonischemic cardiomyopathy in whom clinical findings were consistent with congestive heart failure.

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Methods

Patients. This study was performed with 12 normal volunteers and eight patients with congestive heart failure. The mean ± SD age of the normal subjects was 28 ± 2 years. They were asymptomatic with no evidence of cardiovascular or pulmonary disease on physical examination or two-dimensional echocardiograms. The patients with heart failure were a mean 48 ± 9 years old and had received the following clinical diagnoses: alcoholic (n = 3), postviral (n = 2), hypertensive (n = 1), and idiopathic (n = 2) cardiomyopathy. Patients with ischemic heart disease were not included to avoid the potentially confounding influence of regional wall motion abnormalities. Each of these patients was in sinus rhythm, without conduction abnormalities, had S_2 gallops and bilateral basilar rales, and was clinically judged to be in stable congestive heart failure.

Procedure. The Valsalva maneuver, performed with nose clips, was maintained for 15 sec and standardized at 40 cm of water with a manometer connected to a mouthpiece. Arterial blood pressure was measured with a cuff sphygmomanometer on the left arm of each subject, and the heart rate was determined from a simultaneously recorded electrocardiogram (ECG) during a control period and at the peak of the strain phase, 10 to 15 sec after beginning the Valsalva maneuver. Two-dimensional echocardiograms were obtained continuously in all subjects with the use of a Varian V-3400R echocardiographic system. The subjects were positioned in the left lateral decubitus position and the transducer was placed over the point near the apical cardiac impulse that gave the longest long-axis views of the left ventricle. 11 The apical four-chamber view was obtained from a view transecting the heart from apex to base and perpendicular to the interventricular septum. 12 Images were recorded during a control period while the patients breathed normally and during the late strain phase of the Valsalva maneuver.

Each patient was allowed to fully recover from the first Valsalva maneuver and then the transducer was rotated 90 degrees in a clockwise direction at the same position on the chest wall to obtain the apical two-chamber view. This view transects the heart from apex to base parallel to the interventricular septum. Images were recorded in an identical manner as noted for the four-chamber view during a second control period and during a second Valsalva maneuver. Unfortunately, there was insufficient time during a single Valsalva maneuver to record both apical views.

Images were recorded on a Sanyo VTC 7100 videocassette recorder with vertical and horizontal centimeter calibrations and an ECG was recorded simultaneously. The studies were analyzed by two independent investigators experienced in the technique. Measurements were made during the control period and during the last 5 sec of Valsalva strain. Images were analyzed with the Varian V-3400R microprocessor-controlled video light-pen system and were viewed in real-time, slow-motion, and stop-frame formats. With use of the stop-frame image and with the peak of the R wave defining end-diastole, the right ventricular endocardial border was traced and the area was computed in square centimeters. The left ventricular endocardial borders and the long axis of the left ventricle (apex to the midpoint of the mitral valve annular plane) were drawn with the video light pen. This technique of image processing for the left ventricle was repeated for end-systole, which was defined as the most inward movement of the endocardial image. Left ventricular end-diastolic and end-systolic volumes were calculated independently in both apical views with a modified area-length technique employing programs in the microprocessor unit. The left ventricular stroke volume was calculated as the left ventricular end-diastolic volume minus left ventricular end-systolic volume and ejection fraction was calculated as the stroke volume divided by the end-diastolic volume.

Data analysis. Comparison of data obtained before and during the Valsalva maneuver was performed by a two-tailed paired Student's t test. The level of significance was p < .05. Analysis of the reproducibility of left ventricular volume measurements was done by linear regression analysis.

Results

In each of the normal subjects arterial blood pressure fell during the late strain phase of the Valsalva maneuver and in most it was inaudible by the end of the phase. This fall in blood pressure was associated with an increase in the heart rate from a control value of 68 ± 14 (mean ± SD) to 85 ± 20 beats/min (p < .001). By contrast, the patients with cardiomyopathy and congestive heart failure had an increase in systolic arterial pressure during the strain phase of the Valsalva maneuver from a control value of 118 ± 17 to 131 ± 16 mm Hg (p < .001). The diastolic pressure also increased from the control value of 81 ± 11 to 90 ± 11 mm Hg during the maneuver (p < .05). In these patients heart rate was not significantly different from the control value (90 ± 17 vs 88 ± 18 beats/min).

Sample stop-frame echocardiographic images from a normal subject and a patient are shown in figures 1 and 2. Both groups (figure 3) demonstrated a significant decrease in the right ventricular end-diastolic area during the late strain phase of the Valsalva maneuver compared with control (normal subjects from 9.3 ± 1.5 to 5.6 ± 1.6 cm², p < .001; patients from 13 ± 2.2 to 10 ± 2.9 cm², p < .001).

The consistency of our method of measuring left ventricular volume was assessed by comparing the volumes obtained from each view with results in the same view obtained during the second control period. These data are listed in table 1 and indicate that the measurements were consistent and reproducible.

The response of left ventricular volume to the Valsalva maneuver is shown in table 2 and figures 4 through 7. The normal subjects exhibited a significant decrease in left ventricular end-diastolic volume during the late strain phase of the Valsalva maneuver compared with control that was evident in both the apical four- and two-chamber views (figure 4). The control left ventricular end-diastolic volume in the four-chamber apical view was 96 ± 21 ml and it decreased significantly to 68 ± 18 ml (p < .001) during the Valsalva maneuver. Similarly, the left ventricular end-diastolic volume in the apical two-chamber view decreased from a control level of 97 ± 15 to 56 ± 20 ml (p < .001) during the Valsalva maneuver.

The patients with heart failure did not demonstrate a
significant change in the mean left ventricular end-diastolic volume during the Valsalva maneuver that was apparent in either the apical four-chamber or two-chamber views (figure 5). In the apical four-chamber view, the control left ventricular end-diastolic volume was 199 ± 70 ml as opposed to 195 ± 78 ml (p = NS) during the Valsalva strain. Similarly, in the apical two-chamber view, the control left ventricular end-diastolic volume was 214 ± 77 ml compared with 218 ± 88 ml (p = NS) during the Valsalva period.

In the normal subjects, left ventricular stroke volume decreased significantly in both the apical four-chamber and two-chamber views during the late strain phase of the Valsalva maneuver (figure 6). In the apical four-chamber view, the left ventricular stroke volume during the control period was 64 ± 13 ml compared with 40 ± 11 ml during the Valsalva maneuver (p < .001). In the apical two-chamber view, the left ventricular stroke volume during the control period was 63 ± 9 ml compared with 33 ± 16 ml during the Valsalva period (p < .001).

There was no significant change in the left ventricular stroke volume in the patients from either apical view (figure 7). In the apical four-chamber view the mean left ventricular stroke volume during the control period was 45 ± 21 ml compared with 45 ± 23 ml (p = NS) during the Valsalva period. Likewise, in the apical two-chamber view, the mean left ventricular...
stroke volume for the control period was 49 ± 12 ml as opposed to 49 ± 17 ml (p = NS) during the Valsalva period.

Discussion

The normal response to the Valsalva maneuver has been divided into four phases.3 At the beginning of the maneuver (phase I) the intrathoracic pressure rises abruptly.1–3 This increased pressure is transmitted to the thoracic aorta13 and is reflected as a transient increase in arterial blood pressure.1–3 The increased intrathoracic pressure impedes venous return, resulting in a progressive decline in the right and then left ventricular end-diastolic volumes. This results in a fall in the left ventricular stroke volume and decline in the mean arterial pressure and the pulse pressure (phase II).1–3, 6, 14, 15

This fall in arterial pressure is usually accompanied by a reflexly produced tachycardia and vasoconstriction that serves to maintain mean arterial pressure.1–3 After the release of the Valsalva maneuver (phase III) the venous return abruptly increases, resulting in an increase in stroke volume and a rise or overshoot of the pulse pressure and arterial blood pressure. This normally produces a reflex, parasympathetic slowing of the heart (phase IV). The normal subjects in our study demonstrated this classic response.

More than 40 years ago, Hamilton et al.1 first observed that patients with congestive heart failure had a different response to the Valsalva maneuver. In these patients, the mean arterial pressure and the pulse pressure do not fall during the late strain phase (phase II) of the Valsalva maneuver, but instead remained elevated.1–4 Reflex tachycardia and vasoconstriction are not elicited, and with the release of the maneuver, the blood pressure returns toward normal. The patients with cardiomyopathy and heart failure in our study demonstrated this abnormal response. This pattern, seen in patients with congestive heart failure, has been termed the square-wave blood pressure response and has recently been proposed as a bedside indicator of left ventricular dysfunction.4

Two mechanisms have been proposed to account for this altered response to the Valsalva maneuver in patients with heart failure: (1) The left ventricle may operate on a flat portion of its function curve, which would result in relatively unchanged stroke volume despite a decrease in the left ventricular end-diastolic

TABLE 1

Reproducibility of left ventricular volume measurements

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control</th>
<th>Valsalva</th>
<th>Control</th>
<th>Valsalva</th>
</tr>
</thead>
<tbody>
<tr>
<td>Two-chamber view</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>V1 = 0.97 V2 + 5.4 ml (r = .97)</td>
<td></td>
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<tr>
<td>Four-chamber view</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>V1 = 0.96 V2 + 5.7 ml (r = .96)</td>
<td></td>
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<td></td>
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<tr>
<td>Four-chamber view</td>
<td></td>
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</tr>
<tr>
<td>V4 = 1.08 V3 − 3.25 ml (r = .97)</td>
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</tbody>
</table>

At beginning the Valsalva maneuver vs control 2 (V2)

Edema = left ventricular end-diastolic volume; ESV = end-systolic volume; SV = stroke volume; EF = ejection fraction (SV/EDV).

aP < .001 compared with control.

FIGURE 4. Left ventricular end-diastolic volumes in normal subjects before and during the strain phase of the Valsalva maneuver as determined from apical two-chamber and four-chamber views.

TABLE 2

Response of left ventricular volumes (ml) determined from the four- and two-chamber echocardiograms in normal subjects (n = 10) and patients with cardiomyopathy and congestive heart failure (n = 8)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal subjects</th>
<th>Cardiomyopathy patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Valsalva</td>
</tr>
<tr>
<td>Two-chamber view</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDV 97 ± 15</td>
<td>56 ± 20A</td>
<td>214 ± 77</td>
</tr>
<tr>
<td>ESV 34 ± 11</td>
<td>22 ± 8</td>
<td>165 ± 70</td>
</tr>
<tr>
<td>SV 63 ± 9</td>
<td>33 ± 16A</td>
<td>49 ± 12</td>
</tr>
<tr>
<td>EF 0.67 ± 0.07</td>
<td>0.59 ± 0.10</td>
<td>0.23 ± 0.09</td>
</tr>
<tr>
<td>Four-chamber view</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDV 96 ± 21</td>
<td>68 ± 18A</td>
<td>199 ± 70</td>
</tr>
<tr>
<td>ESV 32 ± 11</td>
<td>28 ± 10</td>
<td>153 ± 59</td>
</tr>
<tr>
<td>SV 64 ± 13</td>
<td>40 ± 11A</td>
<td>45 ± 21</td>
</tr>
<tr>
<td>EF 0.67 ± 0.07</td>
<td>0.59 ± 0.10</td>
<td>0.23 ± 0.09</td>
</tr>
</tbody>
</table>

Edema = left ventricular end-diastolic volume; ESV = end-systolic volume; SV = stroke volume; EF = ejection fraction (SV/EDV).

aP < .001 compared with control.

FIGURE 5. Left ventricular end-diastolic volumes in patients with cardiomyopathy. For details see legend to figure 4.
volume that may result from the decreased venous return during the strain of the Valsalva maneuver.\textsuperscript{2, 3, 5} This effect may be accentuated by a decrease in the effective afterload for left ventricular ejection associated with an increased intrathoracic pressure.\textsuperscript{6-8} (2) The left ventricular end-diastolic volume may be maintained during strain in these patients, in which case stroke volume would not fall because left ventricular preload would be preserved.\textsuperscript{9, 10} This study investigated these two possible mechanisms. In both the normal subjects and in the patients with cardiomyopathy and clinical evidence of congestive heart failure, there was evidence of a marked decrease in systemic venous return during the strain phase of the Valsalva maneuver. The right ventricular end-diastolic size fell in all individuals in both groups. As expected, in the normal subjects by the late strain (phase II) of the Valsalva maneuver the left ventricular end-diastolic and stroke volumes had significantly decreased. In contrast, in the patients with congestive heart failure there was no change in left ventricular end-diastolic or left ventricular stroke volume. This indicates that, despite the decrease in systemic venous return, the filling of the left heart was maintained during the Valsalva strain.

Our findings are consistent with those of previous studies in which M mode echocardiography was used to demonstrate that a single left atrial or left ventricular end-diastolic dimension did not substantially decrease during the strain of the Valsalva maneuver in patients with heart failure.\textsuperscript{9, 10} In these M mode echocardiographic studies shifts in the interventricular septum or other alterations in the configuration of the left ventricle could not be assessed. During other respiratory maneuvers, the position of the interventricular septum may be markedly altered in response to changes in the transseptal pressure gradient.\textsuperscript{16} In our study, the left ventricular volume was separately assessed from the apical two-chamber view, in which the anterior and posterior walls of the left ventricle are imaged, and from the apical four-chamber view, in which the left ventricular lateral wall and the interventricular septum are imaged. Since similar results were obtained from the analysis of both views, indications are that marked changes in the position of the septum are not responsible for the changes that were observed.

In this study, we assessed the effect of venous return on right heart filling by measuring the end-diastolic area of the right ventricle as imaged in the apical four-chamber echocardiographic view. This method was chosen because the geometric complexity of the right ventricle makes determination of its volume difficult. Bommer et al.\textsuperscript{17} demonstrated in studies of right ventricular casts from human hearts that the four-chamber view accurately estimated the dimensions of the right ventricle. They further showed that the area of the right ventricular cavity in this view distinguished patients with right ventricular volume overload from normal individuals. In addition, we showed in a previous study that right ventricular end-diastolic area in this plane correlated with right ventricular end-diastolic counts obtained by gated equilibrium radionuclide angiographic examination of patients with chronic lung disease at rest and after a significant decrease in right ventricular size induced by the administration of sublingual nitrates.\textsuperscript{18} Therefore, we believe that the marked changes in right ventricular area observed during the strain phase of the Valsalva maneuver in our study reflect a true decrease in right ventricular volume.

Left ventricular volumes were estimated in our study by the single-plane area-length ellipsoid geometric approach.\textsuperscript{19} A study of formalin-fixed dog hearts in which this approach was used demonstrated a good correlation with fluid volumes, but underestimation of

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**FIGURE 6.** Left ventricular stroke volumes in normal subjects. For details see legend to figure 4.

**FIGURE 7.** Left ventricular stroke volumes in patients with cardiomyopathy. For details see legend to figure 4.
the actual values. Subsequent studies in intact humans have continued to show good correlations between either the apical two- or four-chamber view and results of cineangiography, as well as consistent underestimation of the actual values for left ventricular volume. No previous study has compared the single-plane volume calculations from each of the apical views. If the ellipsoid geometric assumption is valid, then the values should be the same. Our data from patients at rest (control) showed almost indentical values calculated from the two views and an excellent correlation between them. In addition, the two control values for each view were highly reproducible. During the Valsalva maneuver, the volumes measured in the normal subjects were consistently higher in the four-chamber view. A shift of the interventricular septum toward the markedly smaller right ventricle probably explains this difference. However, the change in volume compared with the control values was in the same direction in both views and the calculated ejection fractions were identical. Unfortunately, a biapical volume calculation approach could not be used because of insufficient time during the late strain phase of the Valsalva maneuver to record two views. We therefore believe that these single-plane left ventricular volume estimates accurately represent the changes in volume induced by the Valsalva maneuver.

This study does not directly address the mechanism by which left ventricular diastolic filling is maintained during Valsalva strain in patients with congestive heart failure. It appears most likely, however, that the increased pulmonary blood volume in these patients serves as a reservoir that maintains left ventricular filling despite the decrease in systemic venous return. Since both the pulmonary veins and the left heart are exposed to intrathoracic pressure, initially a sudden increase in intrathoracic pressure should not change the gradient for diastolic filling of the left heart from the pulmonary veins. The decreased right ventricular volume during the strain phase may shift the left ventricular diastolic pressure-volume relationship downward, facilitating left ventricular filling. In normal subjects, as right heart output falls as a result of the diminished venous return during strain (phase II of the Valsalva maneuver), the pulmonary blood volume is rapidly depleted and left heart filling is decreased. It appears that the increased pulmonary blood volume in patients with congestive heart failure provides for continued left heart filling for a longer period.

In summary, our study demonstrates that in patients with cardiomyopathy and symptoms of congestive heart failure there is a different response of left ventricular volume to the strain phase of the Valsalva maneuver than in normal subjects. In patients with heart failure, despite a decrease in systemic venous return, left ventricular filling is maintained during a 15 sec strain produced by the Valsalva maneuver. This preserved left ventricular end-diastolic volume (preload) appears to be responsible for the maintenance of a normal stroke volume and may explain the abnormal blood pressure response in these patients.

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