Effects of the Valsalva Maneuver on Myocardial Ischemia in Patients with Coronary Artery Disease

CARL J. PEPINE, M.D. AND LESLIE WIENER, M.D.

SUMMARY The influence of the Valsalva maneuver (VM) on myocardial ischemia was evaluated in 24 patients with coronary heart disease. Clinical and hemodynamic responses to the VM were studied during acute ischemia manifested by angina pectoris with transient left ventricular (LV) dysfunction and compared with responses during nonischemic intervals.

In the absence of evidence for acute ischemia (angina and increased LV end-diastolic pressure), six patients had abnormal hemodynamic responses to the VM. Five had lack of systolic pressure overshoot and in one, systolic pressure did not decline during straining. When the VM was performed during an ischemic episode, 14 patients had abnormal responses (12 with lack of overshoot in phase IV and two with lack of systolic pressure decline in phase II). In 18 patients a prompt decline in LV end-diastolic pressure occurred with the disappearance of angina during the VM. These changes uniformly occurred during the latter part of straining (VM phase II) as cardiac size and systolic pressure declined. No adverse effects occurred when a VM was performed during acute ischemia.

Our observations suggest that the VM abruptly reduces determinants of cardiac oxygen demand, relieving acute ischemia without harmful effects.

STRAINING CAUSES exaggerated increases in intrathoracic pressure, frequently during various daily activities. The influence of maneuvers that increase intrathoracic pressure on myocardial ischemia has not been completely determined. For example, the Valsalva maneuver (VM) has been suggested as one of the few nonpharmacologic measures effective in relieving angina pectoris. The basis for this potentially beneficial response on manifestations of ischemia is not completely clear, and others have suggested that this maneuver may cause arrhythmias and "bedpan deaths." These clinical associations were implied because of the important reduction of venous return that occurs during straining, resulting in decreased aortic pressure and cardiac output. Although direct measurements of coronary flow were not made, Gorlin and Storaasli concluded that the VM may reduce blood flow, since it increases the transcoronary circulation time. In a recent study, Benchimol and co-workers implied that the velocity of coronary flow was reduced in patients with and without coronary disease during straining. They estimated unidirectional velocity changes by the Doppler technique, and suggested that reduced flow velocity was secondary to reduced cardiac output and could predispose patients to potentially lethal arrhythmias. If so, improvement in myocardial oxygenation, implied by relief of clinical findings of acute ischemia, is difficult to interpret. Furthermore, the systolic pressure "overshoot" after a VM might induce angina or increase ischemia in some patients. Since this maneuver is performed so frequently, a better understanding of its effects on the coronary circulation may be important.

In the present investigation we studied left ventricular (LV) hemodynamic and clinical responses to a VM in patients with coronary artery disease during acute ischemia. Angina pectoris and a transient rise in LV end-diastolic pressure were considered evidence for ischemia. Emphasis was placed on hemodynamic
changes that occurred during ischemia relating to LV oxygen delivery and demand.

**Methods**

**Patients**

Twenty-four men (mean age 42 years) with easily evoked, typical angina pectoris (New York Heart Association functional class III or IV) were evaluated during diagnostic cardiac catheterization. Each patient had transient ischemic type ST-segment depression (>1 mm) during angina evoked by exercise stress testing or during spontaneous chest pain. None had clinical evidence of congestive heart failure or other forms of cardiac disease. One week before catheterization, adrenergic blocking agents were discontinued. Nitroglycerin was permitted, but only patients who did not use it for at least 2 hours before study were included. Patients were in a fasting, post-absorptive state without premedication, and these studies were performed before angiographic contrast material was administered.

**Catheterization Techniques**

We measured LV pressure during retrograde catheterization with a #7 French NIH catheter. Systemic pressure was measured in 21 patients with a short Teflon catheter (#18 gauge) inserted percutaneously into the left brachial artery. Right atrial pressure (RAP) was obtained from a #7 French Courand catheter inserted through the median antecubital vein.

**Recordings and Calculations**

Systemic, LV and RA pressures were recorded simultaneously from equisensitive Statham P23Db transducers on a multichannel recorder. Mean RAP was obtained by electronic filtration. Recordings were made continuously at 25 mm/sec throughout the maneuvers as described below. Pressures were measured from at least five consecutive beats during the phases outlined. Heart rate was calculated from a standard electrocardiographic lead. Changes in heart size were grossly estimated by a modification of the technique used by Sowton and co-workers. Briefly, cinefluoroscopy was performed before and during the VM (end of phases II and IV) in the posteroanterior projection. From the projected cardiac silhouette, the frontal plane cardiac area at end-diastole was measured. Because of patient-to-patient variation in magnification, the results were expressed as a percentage of change from the pre-VM determination.

**Study Procedure**

Patients were instructed to perform the "classic" VM as a maximal expiratory effort maintained against a closed glottis for 10-12 seconds. They did not try to achieve an arbitrarily set intrathoracic pressure, although all patients maintained at least a 35 mm Hg change in RAP. We observed control VM, performed in an ischemia-free interval, and a VM initiated during transient ischemia. The latter was manifested by angina pectoris associated with an abrupt abnormal increase in LV end-diastolic pressure in each patient. Nineteen of the 24 patients had an ischemic type ST-segment shift during the periods analyzed. These tests were performed as ischemia occurred spontaneously in 12 patients and was evoked by light leg exercise (unloaded bicycle ergometer) in 12 patients. After each VM performed during ischemia, patients were questioned regarding pain relief. Four patients who had used the VM for years to relieve angina, could also signal the precise period during the maneuver when relief began to occur.

Data were obtained from the following phases (fig. 1) for each maneuver: Pre-VM — using 10 beats before each VM. Phase I — at the initial systolic pressure peak, immediately after the onset of strain- ing. Phase II — during plateau, after systolic pressure declined just before release of straining. Phase III — at the maximum systolic pressure decline, immediately after straining release. Phase IV — at the maximum rise or overshoot of systolic pressure after straining release. Post-VM — over the first minute after phase IV. Hemodynamic responses were judged abnormal if the phase IV systolic pressure overshoot was absent without reflex bradycardia or if systolic pressure failed to decline below the pre-Valsalva level during phase II.

After these studies each patient underwent selective coronary and LV angiography. All patients had ≥75% diameter narrowing of at least one major coronary artery. Mean values and standard deviations for each hemodynamic variable were calculated during both the control and ischemic VM. An analysis of variance and t test were used, when appropriate, to examine the statistical significance of the comparisons made.

**Results**

VMs performed during spontaneous angina and stress-induced angina were not different in their clinical or hemodynamic results. Therefore, these data were considered together. Hemodynamic results are summarized in table 1.

**Clinical Responses**

The VM performed during ischemia evoked abnormal responses in 14 of 24 cases, compared with only six during the control VM. Examples of these responses are shown in figures 2-5. Absence of systolic pressure overshoot without bradycardia occurred in 12 of the 14 abnormal responses, five of which were present without transient ischemia. In the other two patients, systolic pressure failed to decline during the ischemic VM, while this response occurred in only one of these two patients during the control VM. Prompt, complete relief of angina resulted in 18 patients. Relief uniformly occurred during the latter part of phase II. One patient had complete relief when systolic pressure...
failed to decline, eight had relief without overshoot, and nine had relief with a normal response. Thus, relief could not be predicted by the type of systolic pressure or heart rate response resulting from the maneuver. When pain was not completely relieved, repeated VMs were effective in producing complete relief in four other patients. No adverse effects such as arrhythmias, syncope, or an increase in intensity of angina occurred during or after the maneuvers. Two patients had occasional ventricular premature depolarizations before catheterization which did not appear to be altered by the VM. The records from one of these patients appear in figure 3.

Hemodynamic Responses

The hemodynamic changes we discuss refer to pre-VM and phase II data from control and ischemic periods, unless noted otherwise (table 1).

**TABLE 1. Hemodynamic Responses to the Valsalva Maneuver During Angina**

<table>
<thead>
<tr>
<th></th>
<th>Pre-VM</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>Post-VM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>78 ± 4</td>
<td>75 ± 5</td>
<td>80 ± 5</td>
<td>85 ± 6</td>
<td>72 ± 4</td>
<td>74 ± 4</td>
</tr>
<tr>
<td>Control VM</td>
<td>85 ± 6</td>
<td>81 ± 5</td>
<td>88 ± 6</td>
<td>89 ± 6</td>
<td>86 ± 6</td>
<td>81 ± 5</td>
</tr>
<tr>
<td>Ischemia VM</td>
<td>2.6 ± 0.6</td>
<td>45 ± 5</td>
<td>51 ± 5</td>
<td>1.1 ± 0.1</td>
<td>4.5 ± 0.7</td>
<td>1.8 ± 0.8</td>
</tr>
<tr>
<td>RAP (mm Hg)</td>
<td>4.0 ± 1.0</td>
<td>43 ± 4</td>
<td>48 ± 5</td>
<td>2.1 ± 0.2</td>
<td>5.0 ± 0.8</td>
<td>3.1 ± 0.7</td>
</tr>
<tr>
<td>Control VM</td>
<td>157 ± 7</td>
<td>192 ± 12</td>
<td>136 ± 6</td>
<td>80 ± 4</td>
<td>173 ± 9</td>
<td>160 ± 8</td>
</tr>
<tr>
<td>Ischemia VM</td>
<td>160 ± 9</td>
<td>198 ± 12</td>
<td>139 ± 6</td>
<td>91 ± 5</td>
<td>170 ± 10</td>
<td>165 ± 8</td>
</tr>
<tr>
<td>LVSP (mm Hg)</td>
<td>11 ± 2</td>
<td>49 ± 4</td>
<td>55 ± 5</td>
<td>4.4 ± 1</td>
<td>14 ± 2</td>
<td>12 ± 2</td>
</tr>
<tr>
<td>Control VM</td>
<td>26 ± 4</td>
<td>64 ± 5</td>
<td>60 ± 5</td>
<td>6 ± 1</td>
<td>18 ± 3</td>
<td>17 ± 3</td>
</tr>
<tr>
<td>Ischemia VM</td>
<td>12,260 ± 675</td>
<td>14,400 ± 768</td>
<td>10,880 ± 510</td>
<td>6800 ± 312</td>
<td>12,456 ± 698</td>
<td>11,840 ± 603</td>
</tr>
<tr>
<td>HR × LVSP (beats/min/mm Hg)</td>
<td>14,368 ± 748</td>
<td>16,045 ± 802</td>
<td>12,232 ± 652</td>
<td>8099 ± 396</td>
<td>14,620 ± 760</td>
<td>13,360 ± 704</td>
</tr>
<tr>
<td>Cardiac area (% change from pre-VM control period)</td>
<td>29 ± 11</td>
<td>4 ± 5</td>
<td>1 ± 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control VM</td>
<td>4 ± 5</td>
<td>1 ± 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemia VM</td>
<td>2 ± 3</td>
<td>3 ± 4</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

N = 24, except cardiac area, where n = 6.

Hemodynamic values represent mean ± sd.

Abbreviations: VM = Valsalva maneuver; RAP = right atrial pressure; LVSP = left ventricular systolic pressure; LVEDP = left ventricular end-diastolic pressure; HR = heart rate.
Effect of VM on Right Atrial Pressure and Heart Rate

With the control VM, mean RAP was lower (2.6 ± 0.6 mm Hg, mean ± sd) and rose to a slightly higher level (51 ± 5 mm Hg) compared with the ischemic VM where mean RAP was higher (4.0 ± 1.0 mm Hg) and increased to 48 ± 5 mm Hg. The pre-VM values were significantly different (2.6 compared with 4.0 mm Hg; \( p < 0.05 \)), but neither phase II values (51 compared with 48 mm Hg) nor the VM-induced pressure change (48 compared with 44 mm Hg) was significant. Heart rate was 78 ± 4 beats/min and increased with the control VM to 80 ± 5 and 85 ± 6 beats/min during phases II and III, respectively. Although there was wide individual variability, heart rate increased in 21 patients during phase II and in 22 of the 24 increased during phase III (\( p < 0.05 \) comparing pre-VM to phases II and III). During ischemia the heart rate was 85 ± 6 (\( p < 0.05 \) compared to control), and no significant change occurred for the group in phases II and III. An increase in rate occurred in only 14 patients during phase II and 16 of the 24 during phase III.

Effect of VM on Left Ventricular Pressures

The control VM was performed at a lower LV systolic pressure than the ischemic VM. (157 ± 7 compared with 169 ± 9 mm Hg; \( p < 0.05 \)). Systolic LV pressure declined significantly (\( p < 0.01 \)) with both maneuvers. Systolic pressures achieved in phase II of both maneuvers were similar (136 ± 6 and 139 ± 6 mm Hg). Likewise, the change (pre-VM and phase II) in systolic pressure with the VM was not significantly different comparing control (13.4%) and ischemic (17.7%) intervals. End-diastolic LV pressure was significantly lower in the control period than during ischemia in each patient studied (11 ± 2 compared with 26 ± 4 mm Hg, \( p < 0.001 \)). End-diastolic pressure was lower during phase II of the control VM than during the ischemic VM in 22 of the 24 patients (55 ± 5 compared with 60 ± 5 mm Hg; \( p < 0.05 \)). The magnitude of LV end-diastolic pressure change during the control VM was also less (\( p < 0.01 \)) than that observed with the maneuver performed during ischemia. The control VM resulted in a 400% increase in end-diastolic pressure, while during ischemia the VM effected only a 130% increase in end-diastolic pressure. End-diastolic pressure declined significantly (\( p < 0.001 \)) in phase III, compared with pre-VM and phase II values during both maneuvers. However, in phase IV and post-VM, end-diastolic pressure declined significantly only during the VM performed during ischemia (18 and 17 mm Hg compared with 26 mm Hg pre-VM; both \( p < 0.05 \)).
Effects of VM on Cardiac Size and Double Product

There was no significant difference between pre-VM control period cardiac area and area measured pre-VM during ischemia. With the control VM, a significantly greater cardiac area decrease occurred during straining compared with the decrease observed during ischemia (29% compared with 14%; p < 0.05). In each of the six patients in whom area was measured, cardiac area decreased less during ischemia than during the control VM. No significant differences in cardiac area measured in phase IV or post-VM, between the control and ischemic VM, were apparent. The double product decreased during phase II of both maneuvers in a similar manner (45% in the control VM and 43% in the ischemic VM, both p < 0.01).

Hemodynamic Effects with Respect to Clinical Response

Analysis of hemodynamic data with respect to clinical responses to the VM yielded findings summarized in figure 6. The subgroup of six patients who did not experience complete relief of angina with the VM had a higher heart rate than the remaining subgroup of patients with relief (pre-VM, 97 ± 5 compared with 80 ± 2 beats/min, p < 0.01). Heart rate during the VM, measured in the patients experiencing pain relief, changed in a manner not significantly different from those without relief. In both patient subgroups LV systolic and end-diastolic pressures were similar before the VM. These pressures, however, were significantly (p < 0.05) higher in the clinically unimproved patient subgroup post-VM, compared with those with improvement. End-diastolic pressure declined in each patient who experienced relief during phase IV and post-VM, compared with pre-VM (14 ± 3 and 14 ± 2 compared with 26 ± 4 mm Hg, both p < 0.05). By contrast, the end-diastolic pressure decline was only minimal and not statistically significant in the subgroup without relief (25 ± 8 and 22 ± 8 mm Hg compared with 28 ± 10 mm Hg). The double product was higher in the subgroup without relief compared with the subgroup with pain relief (16,975 ± 982 compared with 14,400 ± 688, p < 0.05). No significant difference was apparent
Figure 4. Tracings from a patient in whom angina was not relieved with the first of two Valsalva maneuvers (VMs) performed during the ischemic period (top). Systolic pressure declines normally in phase II, but there is lack of systolic pressure overshoot. With a second VM during ischemia, pain was relieved (bottom). With the second VM during ischemia, phase II and IV systolic pressure responses were similar to the previous VM that did not relieve angina (top). A marked reduction in left ventricular end-diastolic pressure occurred with the VM during ischemia. Recordings of right atrial pressure removed for illustration purposes.

in the magnitude of the VM-induced change in double product in phase II between the subgroup without relief compared to those with relief (11 and 13% decrease in phase II). Sufficient data were not available to compare VM effects on cardiac area in the subgroups because area was measured in only two patients without pain relief.

Discussion

The effects of the VM on hemodynamic and clinical responses related to myocardial ischemia are controversial. It has been suggested that a VM-induced increase in intrathoracic pressure may be both beneficial and deleterious. Support for the beneficial effects...
originate from reports of relief of angina pectoris after the VM. The suggestion that it may be harmful derives from studies that implicate the maneuver in the production of bedpan deaths and arrhythmias. Thus, our study was undertaken to determine whether the VM alters either spontaneous or exercise-induced manifestations of ischemia in coronary artery disease patients.

Our results indicate that the VM promptly relieves acute episodes of myocardial ischemia in many of the patients studied, with no harmful effects. Clinical benefit was similar with either spontaneous or effort-evoked angina. To explain relief of ischemia, hemodynamic responses during a VM performed in both ischemic and nons ischemic periods were analyzed. The influence of the VM on ischemia-related increases in LV end-dias tolic pressure supported the subjective angina responses suggesting that the VM relieves manifestations of ischemia. Abrupt end-dias tolic pressure reduction was regularly noted during phase III (immediately after release of straining). The end-

diastolic pressure did fall, in phase III and for several beats into phase IV, in some patients without relief of angina. However, within five to six beats after release of straining, end-diastolic pressure returned to values observed before straining in those who did not have relief. The VM-induced end-diastolic pressure decline persisted throughout phase IV and post-VM periods only in patients reporting relief of angina. Normal heart rate and systolic pressure responses to the VM did not appear to be prerequisites for VM-related improvement. Relief occurred in patients with both normal or abnormal hemodynamic responses. Furthermore, the VM systolic pressure “overshoot” did not induce or increase angina in any patient studied. Most patients observed that symptomatic improvement began during phase II (strain) as the increase in intrathoracic pressure was maintained.

The basis for this salutary effect on transient ischemia was seen in the hemodynamic factors influencing myocardial oxygen requirements and delivery. Important determinants of oxygen requirements include heart rate, myocardial tension and level of contractility. No significant heart rate slowing occurred, possibly indicating an abnormal baroreceptor response. With the VM-induced increase in intrathoracic pressure, LV dimensions are known to remain relatively constant for several beats, while developed pressure declines promptly (table 1). Thus, pressure determinants of wall tension would be expected to decrease almost immediately. During continued straining, systolic and pulse pressures fall as the reduced venous return diminishes ventricular size. With continued increase in airway pressure (phase II), more striking decreases in developed pressure occur, so both pressure and volume determinants of myocardial wall tension decline. The heart rate-systolic pressure product also decreases during straining-induced reduction in venous return. Thus, increases in intrathoracic pressure suddenly reduce some hemodynamic determinants of cardiac oxygen demand.

When evaluating the influence of any agent on myocardial ischemia, one must consider the multiple factors contributing to the relationship of myocardial oxygen demand and supply. With the VM performed during ischemia there was a small increase in heart rate, moderate reduction in systolic pressure and small reduction in cardiac size. Heart size determinations from fluoroscopic tracings are crude estimates, and they were included only as a gross indicator of the directional change in size. During any maneuver that may change the geometry of the heart relative to the thorax, these estimates must be considered cautiously. Similar directional estimates of cardiac size have been reported by others during the VM. Since wall stress is the actual determinant of oxygen consumption, the volume factor is most important and will not be accounted for in the cardiac area or rate-pressure product. Nonetheless, diminished oxygen demand, resulting from VM-related reduction of venous return probably decreases oxygen requirements. This effect would be expected to result in a decrease in coronary flow dur-
ing straining, and may explain some of the prior observations.7, 8

With respect to angina after the VM, comparison of the 18 patients who improved with the six who did not improve (fig. 6) showed that the unimproved patients probably required more oxygen delivery than the improved patients, as evidenced by higher heart rates and systolic pressures. Since no VM-related heart rate decrease occurred in patients with relief, the antianginal response to the VM must relate to other variables. The VM acts to reduce LV size through a reduction in venous return during either spontaneous or effort-evoked ischemia, and thus should reduce oxygen demands. We found lower LV diastolic and reduced systolic pressures in the improved patients, compared with the unimproved patients. This finding may be the result of a greater reduction in venous return and account for relative differences in the hemodynamic effects acting to alter more favorably the myocardial oxygen supply-demand relation. However, we could not be sure whether the decrease in end-diastolic and systolic pressure caused angina relief or resulted from relief by another mechanism.

We did not observe arrhythmias, syncope, dizziness, or other adverse effects. Furthermore, no changes occurred during the period of overshoot to suggest increased ischemia. That intolerance to this procedure was not encountered may relate to the improved hemodynamic function afforded by sudden alleviation of ischemia.

Our observations indicate that the VM promptly relieves manifestations of myocardial ischemia. These studies suggest that the physiologic basis for relief is unrelated to changes in heart rate. Hemodynamic alterations during straining appear to decrease determinants of myocardial tension, reducing cardiac oxygen requirements.

References

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C J Pepine and L Wiener

Circulation. 1979;59:1304-1311
doi: 10.1161/01.CIR.59.6.1304

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